

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE
BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

In Re Application of: William J. Rea and Bertie B. Griffiths

Serial No. 08/902,692

Attorney Docket: 16715CIP

Art Unit: 1644

Filed: July 30, 1997

Examiner: Schwadron, R.

For: **Autogenous Lymphocytic Factor for Modification
of T and B Lymphocyte Parameters**

BRIEF FOR APPELLANTS

Todd E. Albanesi, Reg. No. 36,426
David L. Joers, Reg. No. 31,526
BOOTH ALBANESI SCHROEDER LLC
1601 Elm Street, Suite 1950
Dallas, Texas 75201
(214) 220-0444
Fax: (214) 220-0445
firm@ipoftexas.com

ATTORNEYS FOR APPELLANTS

Table of Contents

I.	REAL PARTY IN INTEREST	2
II.	NO RELATED APPEALS OR INTERFERENCES	2
III.	STATUS OF CLAIMS	2
IV.	STATUS OF AMENDMENTS	2
V.	SUMMARY OF CLAIMED SUBJECT MATTER.....	2
VI.	GROUND OF REJECTION TO BE REVIEWED ON APPEAL	4
VII.	ARGUMENT	4
A.	Grouping of Claims.....	4
B.	Examiner Suggested "Chemically Sensitive"; Objection to Piecemeal Examination.....	4
C.	Summary of New Rejections Under 35 U.S.C. § 112, ¶ 1 (Enablement).....	5
D.	Legal Standards	6
E.	The Examiner Relied on Irrelevant, Unfounded, Prejudicial Allegations	7
F.	The Examiner Failed to Consider Applicants' Evidence	8
G.	The Examiner Misinterprets the Specification	9
H.	"Chemical Sensitivity" Refers to Symptoms, Not to Syndromes or Diseases	9

I.	“Treating” Does Not Mean “Normalizing” the Irregular Cell Cycle for T Lymphocytes	13
J.	Applicants' Evidence of Treating an Irregular Cell Cycle for T Lymphocytes; No Undue Experimentation	15
K.	Conclusion	18
VIII.	CLAIMS APPENDIX.....	20
IX.	EVIDENCE APPENDIX.....	25
X.	RELATED PROCEEDINGS APPENDIX	83

Table of Authorities

CASES

<i>Bristol-Myers Squibb v. Ben Venue Labs., Inc.</i> , 246 F.3d 1368, 58 USPQ2d 1508 (Fed. Cir. 2001)	14
<i>CFMT Inc. v. Yieldup Int'l Corp.</i> , 349 F.3d 1333, 68 USPQ2d 1940 (Fed. Cir. 2003)	14
<i>Fromson v. Advance Offset Plate, Inc.</i> , 720 F.2d 1565, 219 USPQ 1137 (Fed. Cir. 1983)	6
<i>In re Alton</i> , 76 F.3d 1168, 37 USPQ2d 1578 (Fed. Cir. 1996)	8, 9
<i>In re Cortright</i> , 165 F.3d 1353, 49 USPQ2d 1464 (Fed. Cir. 1999)	6, 14
<i>In re Marzocchi</i> , 439 F.2d 220, 169 USPQ 367 (CCPA 1971)	6
<i>In re Sichert</i> , 566 F.2d 1154, 196 USPQ 209 (CCPA 1977)	11
<i>In re Wands</i> , 858 F.2d 731, 8 USPQ2d 1400 (Fed. Cir. 1988)	17
<i>In re Wright</i> , 999 F.2d 1557, 27 USPQ2d 1510 (Fed. Cir. 1993)	6
<i>Kloeris v. Stockdale</i> , 2010 WL 141305 (Tex. App. – Houston [1st Dist.] 2010, pet. denied)	7
<i>Newman v. Quigg</i> , 877 F.2d 1575, 11 USPQ2d 1340 (Fed. Cir. 1983)	6
<i>Rapoport v. Dement</i> , 254 F.3d 1053, 59 USPQ2d 1215 (Fed. Cir. 2001)	11
<i>Scott v. Finney</i> , 34 F.3d 1058, 32 USPQ2d 1115 (Fed. Cir. 1994)	7

OTHER AUTHORITIES

MPEP § 707.07(g)	4
MPEP § 716.01	8
MPEP § 2164.01	16
MPEP § 2164.01(a)	17
MPEP § 2164.03	16
MPEP § 2164.04	6
MPEP § 2164.05	7, 8
MPEP § 2164.08	6

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE
BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

In Re Application of: William J. Rea and Bertie B. Griffiths

Serial No. 08/902,692

Attorney Docket: 16715CIP

Art Unit: 1644

Filed: July 30, 1997

Examiner: Schwadron, R.

For: **Autogenous Lymphocytic Factor for Modification
of T and B Lymphocyte Parameters**

BRIEF FOR APPELLANTS

To: Assistant Commissioner of Patents
Washington, D.C. 20231

Sir:

This is an appeal from the final Office Action mailed January 13, 2011 in the above-identified application for patent.

A Notice of Appeal and fee, together with an Extension of Time Request and fee, was electronically filed via EFS-Web on July 12, 2011.

A copy of the claims involved in the appeal is attached as the Claims Appendix (pp. 20-24).

This brief is also supported by the Declaration of Dr. William J. Rea submitted on September 20, 2010 pursuant to 37 C.F.R. § 1.132. A copy of the Declaration is attached as Exhibit A (pp. 26-82) of the Evidence Appendix.

This Brief for Appellants is accompanied by the requisite fee set forth in 37 C.F.R. § 41.20(b)(2), together with a Petition for Extension of Time Under 37 CFR 1.136(a) and fee, all by electronic filing.

I. REAL PARTY IN INTEREST

The real parties in interest in this application are the named inventors, William J. Rea, Ph.D. and Bertie B. Griffiths, Ph.D., who are referred to herein as the Appellants or Applicants. The application is not assigned.

II. NO RELATED APPEALS OR INTERFERENCES

There are no related appeals or interferences, except that Appellants took two previous appeals in this case to the Board of Patent Appeals and Interferences that were resolved in Appellants' favor before the Board had a chance to rule.

III. STATUS OF CLAIMS

Claims 1–7, 20, 22–31, 33–39, 65–66 and 68–69 have been canceled. Claims 8–19, 21, 32 and 40–48 have been withdrawn from consideration as being drawn to non-elected inventions.

Claims 49–64, 67 and 70 are pending in the application.

Applicants are appealing the final rejection of pending Claims 49–64, 67 and 70.

IV. STATUS OF AMENDMENTS

No amendments have been filed subsequent to the final Office Action mailed January 13, 2011.

V. SUMMARY OF CLAIMED SUBJECT MATTER

Applicants' invention as claimed is directed to "a method for treating a chemically sensitive individual." Although Claims 49–64, 67 and 70 are pending in the application and all the claims do not stand or fall together, Claim 49 is representative as a summary of the invention:

49. A method for treating a chemically sensitive individual having an irregular cell cycle for T lymphocytes, the method comprising the steps of:
- (a) collecting a blood sample from the individual;
 - (b) determining an initial status of the cell cycle for T lymphocytes;

- (c) isolating mixed T and B lymphocytes from the blood sample;
- (d) propagating the isolated mixed T and B lymphocytes to obtain propagated lymphocytes;
- (e) lysing the propagated lymphocytes to obtain a lysate; and
- (f) administering the lysate to the individual.

The claimed method is supported by the specification, including the following general statement in the “Technical Field” section of the specification:

This invention relates to a method of preparing and using an autogenous lymphocytic factor (ALF) for study, modification, and/or regulation of T and B lymphocyte parameters in a mammal, such as a human. The ALF is a substance derived from an individual's own normal T and B lymphocytes isolated from a blood sample and then propagated in a cell culture, which is then administered to the same individual. This invention modulates the abnormal function and levels of the individual's T and B lymphocytes and subsets as measured, for example, by direct flow cytometry, skin sell mediated immunity (CMI) tests, and/or symptoms and signs scores.

Specification at page 1, line 11 – page 2, line 1.

The specification also states (*emphasis added*): “The regulatory effect can be *objectively measured* ... by determining the individual's lymphocytic cell cycle ... and also measured by cell mediated immunity by *skin tests as well as symptoms* and signs scores.” *Id.* at page 11, line 24 – page 12, line 3. The specification states: “Significant changes were observed in patients treated with ALF. Changes were observed in improvement of overall clinical manifestations and immune studies. ... [T]here were significant regulations of lymphocytic cell cycles ... Patients became less sensitive to exposures and more tolerant to specific incitants.” *Id.* at page 14, lines 1–7. The specification states: “The severity of hypersensitive reaction, fatigue, recurrent infections, depression, concentration seemed to improve significantly. ... The frequencies of hypersensitive reaction, recurrent infections, fatigue, headaches, and depression were also altered.” *Id.* at page 16, lines 19–22.

VI. GROUNDS OF REJECTION TO BE REVIEWED ON APPEAL

The grounds of rejection to be reviewed on appeal are: whether Claims 49–64, 67 and 70 are unpatentable under 35 U.S.C. § 112, ¶ 1, as failing to comply with the enablement requirement.

VII. ARGUMENT

A. Grouping of Claims

Appellants state that all the claims do not stand and fall together, and request the following grouping of claims.

Claims 49–64, 67 and 70 are currently pending in the application. Claims 49, 60 and 70 are the only independent claims.

Claims 49 and 60 include the preamble language “A method for treating a chemically sensitive individual having an irregular cell cycle for T lymphocytes.” Independent Claim 60 is a more specific claim.

The preamble of Claim 70 does not include the language “having an irregular cell cycle for T lymphocytes.”

Regarding Claims 49–64 and 67, the specification provides evidence of enablement for use in treating certain individuals: those who are chemically sensitive and have an irregular cell cycle for T lymphocytes. Regarding Claim 70, the specification provides evidence of enablement for use in treating those who are chemically sensitive.

Accordingly, for the limited purposes of this appeal, Applicants group and will argue the pending claims as follows:

Group I: Claims 49–64 and 67 stand or fall together, and Applicants suggest that Claim 49 is representative of this group;

Group II: Claim 70 stands or falls separately.

B. The Examiner Suggested “Chemically Sensitive”; Objection to Piecemeal Examination

The piecemeal examination of this application is contrary to the policy of the Patent and Trademark Office. MPEP § 707.07(g).

The independent claims were amended nearly 13 years ago to be directed to a method for treating “a chemically sensitive individual,” *which was originally suggested by the Examiner in a telephone conference on March 16, 1999, and which had been in the claims ever since.* See the Amendment filed April 17, 1999, page 4. (The claims as originally filed were directed to methods for regulating an “*abnormal lymphocytic cell cycle*” (claims 1–39) and for treating an individual “*having abnormal T and B lymphocyte parameters*” (claims 40–48)).

For the first time in 13 years, this language is used as the basis of a new rejection. This is after numerous rejections on various other piecemeal grounds, all of which Applicants have overcome, including by making two different prior appeals to the Board of Patent Appeals and Interferences.

C. Summary of New Rejections Under 35 U.S.C. § 112, ¶ 1 (Enablement)

The 1/13/2011 final Office Action rejected the claims under 35 U.S.C. § 112, ¶ 1, as failing to comply with the enablement requirement. The Examiner did not reject the claims for lack of utility or on the basis of prior art.

In outline form, the new rejection is that the application does not “enable” a person of skill in the art to make and use the claimed invention, including the following arguments:

*“chemical sensitivity” is not a defined diagnosis and cites an anonymous complaint filed with the Texas Medical Board questioning the validity of the diagnosis;

*the claimed invention encompasses the treatment of a “plethora” of diseases;

*the state of the art is “unpredictable”;

*the evidence of Figures 2–4 is unclear;

*there is no “appropriate control group”;

*the concentration of any protein from ALF would be below that used for any biological modifier to treat humans;

*the description does not teach the full scope of the claimed invention without “undue experimentation.”

D. Legal Standards

To be enabling, the specification of a patent must teach those skilled in the art how to make and use the full scope of the claimed invention without “undue experimentation.” *In re Wright*, 999 F.2d 1557, 1561, 27 USPQ2d 1510, 1513 (Fed. Cir. 1993). The “invention” that must be enabled is that defined by the particular claim or claims of the patent or patent application. *See* MPEP § 2164.08 (“All questions of enablement are evaluated against the claimed subject matter.”).

It is not a requirement of patentability that an inventor correctly set forth, or even know, how or why the invention works. *Newman v. Quigg*, 877 F.2d 1575, 1581, 11 USPQ2d 1340, 1345 (Fed. Cir. 1983). An inventor need not comprehend the scientific principles upon which the practical effectiveness of the invention rests. An expression of theory merely as a belief will not be used to limit the claims; indeed, § 112 does not require a statement of theory or belief. *Fromson v. Advance Offset Plate, Inc.*, 720 F.2d 1565, 1570, 219 USPQ 1137, 1140 (Fed. Cir. 1983). “[S]tatements that a physiological phenomenon was observed are not inherently suspect simply because the underlying basis for the observation cannot be predicted or explained.” *In re Cortright*, 165 F.3d 1353, 1359, 49 USPQ2d 1464, 1469 (Fed. Cir. 1999).

To make a rejection, the examiner has the initial burden to establish a reasonable basis to question the enablement provided for the claimed invention. *In re Wright*, 999 F.2d 1557, 1562, 27 USPQ 2d 1510, 1513 (Fed. Cir. 1993); MPEP § 2164.04. Assuming that sufficient reason for such doubt does exist, a rejection for failure to teach how to make and/or use will be proper on that basis; such a rejection can be overcome by suitable proofs indicating that the teaching contained in the specification is truly enabling. *In re Marzocchi*, 439 F.2d 220, 224, 169 USPQ 367, 370 (CCPA 1971). When a rejection is made on the basis that the disclosure lacks enablement, it is incumbent upon the Examiner to explain why he/she doubts the truth or accuracy of any statement in a supporting disclosure and to back up assertions with acceptable evidence or reasoning which is inconsistent with the contested statement. *Id.*

E. The Examiner Relied on Irrelevant, Unfounded and Prejudicial Allegations

In the 1/13/2011 final Office Action, on page 3, the Examiner states:

... Barrett (2007) discloses a complaint filed against Inventor Rea filed with the Texas Medical Board which questions the validity of the diagnosis and treatment of chemical sensitivity as proffered by Inventor Rea. ...

These allegations are irrelevant, unfounded and prejudicial and should not be considered. 9/20/2010 Declaration of William J. Rea, ¶¶ 12–20, Ex. A, pp. 27–28.

On August 27, 2010, the referenced complaint was resolved by Mediated Agreed Order (a settlement agreement). 9/20/2010 Declaration of William J. Rea, ¶ 14, Ex. A, p. 27. The Agreed Order is a settlement agreement under the Rules of Evidence. *Id.* at p. 4, ¶ 5; *Kloeris v. Stockdale*, 2010 WL 141305, at *9 (Tex. App. – Houston [1st Dist.] 2010, pet. denied) (same; settlement agreements are not admissible to prove liability).

The matter concerned different treatment methods (use of chemical antigens, oxygen treatments, etc.) not at issue in the present application for patent or the claims. See the Agreed Order, at p. 3, ¶ 3; see also 9/20/2010 Declaration of William J. Rea, ¶ 16, Ex. A, p. 28. This was not an adjudication or findings. 9/20/2010 Declaration of William J. Rea, ¶ 18, Ex. A, p. 28.

To settle that matter, Dr. Rea agreed to change the Informed Consent documents used in his medical practice. 9/20/2010 Declaration of William J. Rea, ¶¶ 12–20, Ex. A, pp. 27–28.

In addition, regarding the settlement Informed Consent documents, it is not required that a method, especially a medical treatment help in every case. “Considerations made by the FDA for approving clinical trials are different from those made by the PTO in determining whether a claim is enabled.” MPEP § 2164.05, citing *Scott v. Finney*, 34 F.3d 1058, 1063, 32 USPQ2d 1115, 1120 (Fed. Cir. 1994) (“Testing for full safety and effectiveness of a prosthetic device is more properly left to the [FDA].”).

In the 1/13/2011 final Office Action, on pages 2–3, the Examiner also states:

... In addition, regarding Inventor Rea and the use of the factor recited in the claims (aka ALF aka autogenous lymphocytic factor),

Hall indicates that is unclear if ALF can actually be used to treat disease (see pages 3–4). ...

The reference to Hall (2009) is irrelevant and prejudicial and should not be considered. 9/20/2010 Declaration of William J. Rea, ¶¶ 21–25, Ex. A, pp. 28–29. Hall (2009) does not address the claimed subject matter in the present application for patent. *Id.* ¶ 23. Hall (2009) references a television “profile” on “Nightline” in 2008. Hall (2009), p. 2. Nightline is a popular television news show, not any kind of a scientific forum. *Id.* ¶ 24.

F. The Examiner Failed to Consider Applicants’ Evidence

Applicants submitted the September 20, 2010 Declaration of Dr. William J. Rea, Ex. A, in response to the 3/18/2010 non-final Office action. However, the Examiner ignored Applicants’ evidence. The Examiner essentially repeated the prior rejection. Pages 2–6 of the 1/13/2011 final Office Action appear to be a verbatim copy of the statements in the previous Office Action. The general statement “Applicants’ arguments have been considered and not deemed persuasive” was inserted before repeating the same arguments. See Paragraph 3 on page 2.

On page 6, the second to last paragraph, the phrase “Regarding applicants comments ...” (*was substituted* for the previous “Regarding Wands factors 4–8” language), and then, rather than saying anything different, it essentially repeats *again*, apparently verbatim, the text of pages 2–6 at pages 7–10. The only difference between the two repeated sections appears to be the addition of two paragraphs of legal citations, one bridging pages 6–7 and the other in the middle of page 8, each paragraph including a citation to the MPEP and a few related legal cases. The Examiner did not respond to a single factual statement made in Dr. Rea’s Declaration, Ex. A, or make any specific mention of it at all.

Applicant’s evidence ***must*** be considered. A declaration or affidavit is, itself, evidence that ***must*** be considered. MPEP § 2164.05. The examiner must then weigh all the evidence before him or her, including the specification and any new evidence supplied by applicant and decide whether the claimed invention is enabled. *Id.* See MPEP § 716.01: “Evidence traversing rejections must be considered by the examiner whenever present. ... Where the evidence is insufficient to overcome the rejection, the examiner must specifically explain why the evidence is insufficient.” See also *In re Alton*, 76 F.3d 1168, 1174, 37 USPQ2d 1578, 1583 (Fed.

Cir. 1996) (declarations relating to the written description requirement should have been considered).

G. The Examiner Misinterprets the Specification

The specification never asserts that chemical sensitivity “causes” or “is linked to” a “plethora” of diseases as the Examiner implies.

The Examiner states “[t]he specification has not enabled the breadth of the claimed invention ... because the use for the claimed methods is the in vivo treatment of a *plethora of disease* apparently *linked to chemical sensitivity* in humans.” 1/13/2011 final Office Action, p. 3. (Emphasis added).

The Examiner refers to “the various diseases *which the specification links to ‘chemical sensitivity’ (pages 1–4)*” (1/13/2011 final Office Action, p. 2, last ¶, 3 lines from the bottom); and “a wide variety of diseases ‘*caused by chemical sensitivity*’” (*id.*, p. 3, first full ¶, second line). Applicants cannot find where this proposition is supposedly asserted in the specification. Indeed, the phrase “caused by chemical sensitivity” is apparently purportedly quoted from the specification, but is not found in the specification.

Contrary to the Examiner’s implication, the specification states (at page 4, lines 6–20) (*emphasis added*):

[T]he application of this invention is useful in the study of the immune system, and ... is not limited to the treatment of a certain category of individuals. For example, the method can be applied to the study and/or clinical treatment of individuals suffering from a *suppressed, dysfunctional, or deregulated immune system for any number of possible causes*. However, the emphasis of this invention is on the treatment of the individuals who have *compromised immune systems* that result in an *abnormal susceptibility to environmental chemicals (chemically sensitive)*...

H. “Chemical Sensitivity” Refers to Symptoms, Not to Syndromes or Diseases

In the 1/13/2011 final Office Action (at pp. 2–3), the Examiner states (*emphasis added*):

... the term ‘chemically sensitive individual’ is not specifically defined in the specification. However, based on page 12, last paragraph, and page 15, last paragraph of the specification said

patients apparently encompass those suffering from dermatitis, vasculitis, asthma, organic brain syndrome, gulf war syndrome or immune system specific suppression, dysfunction or deregulation and arthritis. However, Orme et al. indicates that it is unclear if a diagnostic entity such as the '*chemically sensitive individual*' (*aka multiple chemical sensitivity*) with the aforementioned diseases actually occurs ...

The Examiner also states (at p. 3):

The claimed invention encompasses the treatment of a wide variety of diseases 'caused by chemical sensitivity' including patients suffering from dermatitis, vasculitis, asthma, organic brain syndrome, gulf war syndrome or immune system specific suppression, dysfunction or deregulation and arthritis. The aforementioned collection of diseases would encompass a plethora of autoimmune and inflammatory diseases."

Contrary to the Examiner's new interpretation, "chemical sensitivity" or "chemically sensitive individual" refers to symptoms. 9/20/2010 Declaration of William J. Rea, ¶¶ 27–31, Ex. A, pp. 29-30. The specification defines and uses "chemically sensitive" referring to symptoms. 9/20/2010 Declaration of William J. Rea, ¶¶ 32–35, Ex. A, p. 31.

The specification (at page 4, lines 10–14) defines how the term "chemically sensitive" is being used in the specification and claims (*emphasis added*):

... However, the emphasis of this invention is on the treatment of the individuals who have compromised immune systems *that result in an abnormal susceptibility to environmental chemicals (chemically sensitive)*, pollens, dust, molds, food (allergies), bacteria and non-HIV viruses with recurrent infections.

In addition, the specification (at page 13, lines 16–19) identifies this principal characteristic of "chemically sensitive individuals":

The total of 290 chemically sensitive individuals that were investigated in these studies were affected principally by environmental incitants found in categories such as food, biological inhalants, and chemicals. They presented histories of varied backgrounds, but common among them was that all showed irregular cell cycles including T and B lymphocytes ...

The term “chemically sensitive” individual is defined and used in the specification and claims with reference to *symptoms, signs and abnormal laboratory data*.

The application does *not* use the term “multiple chemical sensitivity.” The claims are *not* directed to “multiple chemical sensitivity” (aka “MCS”). 9/20/2010 Declaration of William J. Rea, ¶ 36, Ex. A, p. 31. “Multiple chemical sensitivity” (“MCS”) refers to a syndrome. 9/20/2010 Declaration of William J. Rea, ¶¶ 37–40, Ex. A, p. 32. “Chemical sensitivity” symptoms are *not* “multiple chemical sensitivity” syndrome and should not be equated. 9/20/2010 Declaration of William J. Rea, ¶ 41, Ex. A, pp. 32-33. The claims are *not* directed to other diseases. 9/20/2010 Declaration of William J. Rea, ¶¶ 33–34, Ex. A, p. 31.

The Examiner’s interpretation is contrary to the specification and the record as a whole. *See In re Sichert*, 566 F.2d 1154, 1160–61, 196 USPQ 209, 214 (CCPA 1977) (in patent application for therapeutical compositions for treating lymphatic congestions, rejection for lack of enablement was improper, since *there was no need for a disclosure* as to which diseases are encompassed by the term “lymphatic congestions” *when the meaning of that term* was limited to simple lymphatic congestion (ellogged lymph vessels) and did not extend to the causes or results of lymphatic congestion (various therapy resistant diseases); “The record clearly shows that the compositions were developed for the ‘activation and regeneration of the lymphatic vessels ...; that one of the *criteria for determining the effectiveness* of the composition is the ‘tendency of the swellings of the tissues to decrease ...; that these compounds are ‘suited to diminish inflammatory changes in the lymphatic system ...; and that the result of treatment is ‘the effective drainage of the entire lymphatic system.’”). For the reverse situation *see Rapoport v. Dement*, 254 F.3d 1053, 1058–60, 59 USPQ2d 1215, 1219–20 (Fed. Cir. 2001) (senior party’s claims were not anticipated, since term “treatment of sleep apneas” in patent interference count referred only to treatment of underlying sleep disorder itself and not to treatment of secondary symptoms; *ordinary meaning* of term referred to treatment of underlying disorder, written description defined “sleep apneas” in terms of underlying disorder, and *description of efficacy of claimed treatment method* only addressed its effect on underlying disorder).

Here, the specification describes the efficacy of the claimed method in terms of its effect on symptoms.

For example, the specification states (at page 11, line 22 – page 12, line 3):

Initial clinical testing indicates that a first treatment ... can have a significant effect on improving the individual's T and B lymphocyte parameters. The regulatory effect can be objectively measured ... by determining the individual's lymphocytic cell cycle ... and also measured by cell mediated immunity by skin tests as well as symptoms and signs scores.

The specification also states (at page 14, lines 1–7):

Significant changes were observed in patients treated with ALF. Changes were observed in improvement of overall clinical manifestations and immune studies. ... [T]here were significant regulations of lymphocytic cell cycles ... Patients became less sensitive to exposures and more tolerant to specific incitants.

The specification states (at page 16, lines 19–22):

The severity of hypersensitive reaction, fatigue, recurrent infections, depression, concentration seemed to improve significantly. ... The frequencies of hypersensitive reaction, recurrent infections, fatigue, headaches, and depression were also altered.

The specification explains that: “[T]he hypersensitivity reactions markedly decreased or disappeared. ... [R]ecurrent infection, fatigue, headaches, depression, concentration, even gastrointestinal upsets were also improved.” *Id.* at page 18, lines 21–24.

The specification states: “Symptoms and signs scores are seen in TABLE 15 and TABLE 16. Significant improvement occurred ... The severity of hypersensitive reaction, fatigue, recurrent infections, depression, concentration seemed to improve significantly. The frequencies of hypersensitive reaction, recurrent infections, fatigue, headaches, and depression were also altered.” *Id.* at page 20, lines 16–20. The specification also states: “TABLE 22 shows a case study of one ... HIV-positive patient ... After restarting the ALF treatment, all her clinical symptoms disappeared again.” *Id.* at page 22, lines 6–12.

I. “Treating” Does Not Mean “Normalizing” the Irregular Cell Cycle for T Lymphocytes

The 1/13/2011 final Office Action overstates the scope of the claimed invention, on page 3 (*emphasis added*):

Regarding the use of the claimed method to regulate an abnormal lymphocytic cell cycle of continuously dividing *T and B lymphocytes* in a mammal wherein said method encompasses the in vivo treatment of humans, there is no evidence in the specification that such regulation has been achieved using the claimed method. Regarding Wands factors 1–3, the claimed method encompasses a method wherein according to the specification the abnormal lymphocytic cell cycle of continuously dividing *T and B lymphocytes in a mammal is normalized*.

Pending claims 49–64 and 67 are directed to “A method for treating a chemically sensitive individual having an irregular cell cycle for T lymphocytes” Claim 70 is similar to pending Claim 49 *except* that the preamble does *not* include the language “having an irregular cell cycle for T lymphocytes.”

In addition, the specification and claims do not require the irregular cell cycle for T lymphocytes be “*normalized*.” Of course, this is a desired goal, but the claims are directed to “*treating*,” which is illuminated by the specification:

... treating the individual with a therapeutic amount of the ALF, and determining the individual's lymphocytic cell cycle to observe any regulatory effect on the lymphocytic cell cycle and subsets.

Specification, page 6, lines 16–18.

... As treatment [with ALF] continued, in general, in about six weeks a more drastic shift *toward that of a normal profile was observed.*

Specification, page 14, lines 8–9 (*emphasis added*).

It is for the invention *as claimed* that enablement must exist. The term “normalizing” does not appear in the claims. The claims state no standard of regulation. The application and claims do not require complete regulation, but is *a basis* for regulation of the cell cycle. 9/20/2010 Declaration of William J. Rea, ¶¶ 47–49, Ex. A, pp. 34–35. See *CFMT Inc. v. Yieldup Int’l Corp.*, 349 F.3d 1333, 1338, 68 USPQ2d 1940, 1944 (Fed. Cir. 2003) (when an invention claims a general system to improve the cleaning process for semiconductor wafers, the disclosure enables that invention by showing improvements in the overall system; if a patent claimed a system that achieved cleanliness up to a specified numerical particle-free range, then enablement would require disclosure of a method that enables one of ordinary skill to achieve that range without undue experimentation; “The claims of the ‘532 and ‘123 patents *state no standard of cleaning. ... ‘[C]leaning in the context of this invention means generally removing contaminants from the wafer surface.*” (Emphasis added.)); *In re Cortright*, 165 F.3d 1353, 1359, 49 USPQ2d 1464, 1468 (Fed. Cir. 1999) (“[O]ne of ordinary skill *would not construe ‘restoring hair growth’ to mean ‘returning the user’s hair to its original state,* as the board required. To the contrary, consistent with Cortright’s disclosure and that of other references, one of ordinary skill would construe this phrase as meaning that the claimed method increases the amount of hair grown on the scalp, but does not necessarily produce a full head of hair. Properly construed, claim 1 is amply supported by the written description because Example 1 discloses the amount of Bag Balm® to apply (about one teaspoon daily) and the amount of time (about one month) in which to expect results. These dosing instructions enable one of ordinary skill to practice the claimed invention without the need for any experimentation.” (Emphasis added.)) See also, *Bristol-Myers Squibb v. Ben Venue Labs., Inc.*, 246 F.3d 1368, 1375–76, 58 USPQ2d 1508, 1513 (Fed. Cir. 2001) (preamble language “for reducing hematologic toxicity” was “non-limiting, and merely express[ed] a purpose of reducing hematological toxicity ... The steps of the ... method are performed in the same way regardless whether or not the patient experiences a reduction in hematologic toxicity ...”).

J. Applicants' Evidence of Treating an Irregular Cell Cycle for T Lymphocytes; No Undue Experimentation

The Examiner complains that the data in Figures 2–4 “provides no information about the cell cycle of human peripheral B lymphocytes from ‘normal’ volunteers.” (1/13/2011 final Office Action, p. 4). But the normal cell cycle for both T and B lymphocytes was well known at the time the invention was made and the application was filed. 9/20/2010 Declaration of William J. Rea, ¶¶ 50–56, Ex. A, pp. 35–36. The specification evidences improvement in the cell cycle for T lymphocytes. 9/20/2010 Declaration of William J. Rea, ¶¶ 57–67, Ex. A, pp. 36–39. In the 1/13/2011 final Office Action, on page 4, the Examiner continues: “Figures 3a–3c purport to show the ‘irregular cell cycle profiles from environmentally compromised individuals.’ There is no disclosure as to what cells are referred to in said figure ...” However, it is clear from the specification that unless otherwise specified, the “cell cycle” refers to the cell cycle for mixed T and B lymphocytes, including all subsets, which is disclosed to present a reflection of the status of the T lymphocytes, including all subsets. No other interpretation is reasonable. 9/20/2010 Declaration of William J. Rea, ¶ 61, Ex. A, p. 37.

Among other evidence provided in the specification, the written description states:

Significant changes were typically observed in patients treated with ALF. Changes were observed in improvement of overall clinical manifestations and immune studies. With regard to clinical manifestations, minimal symptoms (which were improved over the onset symptoms) continued after three weeks of continued therapy with ALF. Immunologically, there were significant regulations of cell cycles, especially from one phase of the cycle to another, and changes in T and B lymphocyte numbers and functions. Patients became less sensitive to exposures and more tolerant to specific incitants. As treatment continued, in general, in about six weeks a more drastic shift toward that of a normal profile was observed.

Specification, page 14, lines 1–8 (*emphasis added*).

The Examiner also states (p. 4) that “it appears that the cell cycle of untreated patients in Figure 4a more closely approximates that seen in the normal controls” Figures 4a–c represent *a single case history*. These figures *illustrate* to a person of skill in the art a drastic improvement

in the cell cycle, which is a reflection of the improvement of the cell cycle for T lymphocytes. . 9/20/2010 Declaration of William J. Rea, ¶ 67, Ex. A, pp. 38-39.

In addition, the claims do not require regulation of the cell cycle in patients suffering from autoimmune disease. 9/20/2010 Declaration of William J. Rea, ¶¶ 68–70, Ex. A, p. 39.

The results obtained are too large to attribute to the placebo effect or any of the other therapies or treatments that had been used until that time in environmental medicine. 9/20/2010 Declaration of William J. Rea, ¶¶ 71–72, Ex. A, pp. 39-40. In addition, a theoretical explanation was offered in the specification. 9/20/2010 Declaration of William J. Rea, ¶¶ 73–74, Ex. A, p. 40.

Under the Wands factors, no undue experimentation would be required. Preliminary evidence was provided at the time of filing the application of some highly successful treatments according to the claimed invention, in rate of response, degree of response, and frequency of response that could only be attributed to the new treatment with ALF. The specification provides a “cook book” example of the procedure for practicing the invention. The statements contained in the written description regarding the scope of the claimed invention as set forth in the presently pending claims are supported by the data presented. The level of the skill regarding the subject matter of the claimed invention is high. Based on the description in the specification and the above factors, there is no requirement for “undue experimentation.” A person of skill in the art, based on the invention disclosure and with good financial and time resources, could conduct additional testing and clinical trials using the invention to elucidate the cause-and-effect relationships involved. 9/20/2010 Declaration of William J. Rea, ¶¶ 75–79, Ex. A, pp. 40-41.

The enablement requirement of § 112 is satisfied when an application describes a claimed invention in a manner that permits one of ordinary skill in the art to practice it, without undue experimentation. MPEP § 2164.01. Thus, the mere fact that experimentation might be required is insufficient to support an enablement rejection. Further, even complex experimentation is not necessarily undue. MPEP § 2164.01.

Even if experimentation may be required in this case, it would not be undue. The question of enablement is one of predictability in view of what is known in the art. Consequently, the amount of guidance or direction needed to satisfy the enablement requirement is inversely related to the amount of knowledge in the state of the art as well as the predictability in the art. MPEP § 2164.03.

The specific question of whether experimentation is “undue” is determined based on the following eight *Wands* factors:

1. Breadth of the claims;
2. Nature of the invention;
3. State of the prior art;
4. Level of ordinary skill in the art;
5. Predictability of the art;
6. Amount of direction provided in the specification;
7. Any working examples; and
8. Quantity of experimentation needed relative to the disclosure.

MPEP § 2164.01(a), citing *In re Wands*, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988). Further, a proper analysis of whether any experimentation is undue requires an analysis of *all* of the pertinent *Wands* factors. MPEP § 2164.01(a) (emphasis added). It is improper to conclude that a disclosure is not enabling based on an analysis of only one of the above factors while ignoring one or more of the others. *Id.*

The Applicants’ preliminary work adequately establishes that the methods would have application beyond the specific work reported in the specification. A patent is not required to rise to the level of testing required for new drug approval or drug efficacy claims, but must be merely sufficient to teach to a person of ordinary skill in the art how to make and use the invention without undue experimentation. Based on the Applicants’ specification, a person of skill in the art would certainly be able to appreciate the invention and conduct any further routine studies that may be desirable, including regarding the cell cycles of any of the lymphocytes, determine an initial status of a lymphocyte cell cycle of the individual, make ALF according to the method, administer the ALF to the individual, and observe the effects on the cell cycle of the individual’s lymphocytes, all according to the teaching of the specification and as claimed.

In view of the foregoing, Applicants respectfully submit that a person of ordinary skill in the art would be able to make and use the claimed invention, despite any experimentation that might be required. Applicants further submit that this conclusion is buttressed by the amount of knowledge in the state of the art as well as the predictability of the art, as well as the majority of

Wands factors that weigh in favor of enablement. Therefore, the present application adequately enables the claimed invention.

K. Claim 70 Does Not Recite “Having an Irregular Cell Cycle for T Lymphocytes”

Claim 70 is argued separately only to point out there is even less justification for reading a “normalizing” standard into Claim 70 (subhead I, *supra*).

Claim 70 is similar to pending Claim 49 except that the preamble does not include the language “having an irregular cell cycle for T lymphocytes.”

Regarding Claims 49–64 and 67, the specification provides evidence of enablement for use in treating certain individuals: those who are chemically sensitive and have an irregular cell cycle for T lymphocytes. Regarding Claim 70, the specification provides evidence of enablement for use in treating those who are chemically sensitive.

As discussed (above subhead I), “treating,” properly construed, does not mean “normalizing” the irregular cell cycle for T lymphocytes. This interpretation is even more compelling regarding Claim 70, which does not recite “having an irregular cell cycle for T lymphocytes.”

L. Conclusion

Based on the foregoing evidence, arguments, and authorities, it is respectfully requested that the rejection of pending Claims 49–64, 67 and 70 under 35 U.S.C. § 112, first paragraph, be reversed and the application be allowed for issue.

Dated: February 13, 2012.

Respectfully submitted,

/s/ Todd E. Albanesi

Todd E. Albanesi, Reg. 36,426
BOOTH ALBANESI SCHROEDER LLC
1601 Elm Street, Suite 1950
Dallas, Texas 75201-4744
(214) 220-0444
Fax: (214) 220-0445

ATTORNEYS FOR APPELLANTS

VIII. CLAIMS APPENDIX

- 1-7. Canceled.
- 8-19. Withdrawn.
- 20. Canceled.
- 21. Withdrawn.
- 22-31. Canceled.
- 32. Withdrawn.
- 33-39. Canceled.
- 40-48. Withdrawn.

49. (Previously Amended) A method for treating a chemically sensitive individual having an irregular cell cycle for T lymphocytes, the method comprising the steps of:

- (a) collecting a blood sample from the individual;
- (b) determining an initial status of the cell cycle for T lymphocytes;
- (c) isolating mixed T and B lymphocytes from the blood sample;
- (d) propagating the isolated mixed T and B lymphocytes to obtain propagated lymphocytes;
- (e) lysing the propagated lymphocytes to obtain a lysate; and
- (f) administering the lysate to the individual.

50. (Previously Presented) The method according to Claim 49, wherein the step of collecting a blood sample further comprises the step of: collecting the blood sample from the individual by venipuncture in heparinized tubes.

51. (Previously Amended) The method according to Claim 49, wherein the step of isolating mixed T and B lymphocytes from the blood sample further comprises the steps of: separating the erythrocytes and neutrophils from the lymphocytes of the blood sample by a sodium diatrizoate and polysucrose density gradient technique to obtain a lymphocytic sample; centrifuging the lymphocytic sample; separating and combining the lymphocytic layers from the

centrifuged lymphocytic sample; and washing the combined lymphocytic layers to obtain the isolated mixed T and B lymphocytes.

52. (Previously Presented) The method according to Claim 49, wherein the step of propagating the isolated mixed T and B lymphocytes further comprises the steps of: culturing the isolated mixed T and B lymphocytes with a cell growth medium at about 37°C.

53. (Previously Presented) The method according to Claim 52, wherein the cell growth medium is supplemented with bovine calf serum.

54. (Previously Presented) The method according to Claim 52, wherein the step of propagating the lymphocytes further comprises the steps of: centrifuging the cultured lymphocytes; removing the supernate from the centrifuged lymphocytes; and washing the centrifuged lymphocytes in normal saline with further centrifugation to obtain the propagated lymphocytes.

55. (Previously Presented) The method according to Claim 49, wherein the step of lysing the propagated lymphocytes further comprises the steps of: suspending the propagated lymphocytes in normal saline solution; sonicating the suspended lymphocytes; and filtering the sonicated lymphocytes to obtain the lysate.

56. (Previously Presented) The method according to Claim 49, wherein the step of administering the lysate to the individual further comprises the step of: determining a therapeutic dose of the lysate by skin testing.

57. (Previously Presented) The method according to Claim 56, wherein the step of administering the lysate to the individual comprises the step of: injecting the individual subcutaneously with the therapeutic dose of the lysate.

58. (Previously Presented) The method according to Claim 57, further comprising the step of: injecting the individual subcutaneously with at least one additional therapeutic dose of the lysate.

59. (Previously Presented) The method according to Claim 49, further comprising the steps of: measuring the clinical symptoms and signs of the individual before administering the lysate, and then measuring clinical symptoms and signs of the individual after administering the lysate.

60. (Previously Amended) A method for treating a chemically sensitive individual having an irregular cell cycle for T lymphocytes, the method comprising the steps of:

- (a) collecting a blood sample from the individual by venipuncture in heparinized tubes;
- (b) determining an initial status of the cell cycle for T lymphocytes;
- (c) isolating mixed T and B lymphocytes from the blood sample by:
 - (i) separating the erythrocytes and neutrophils from the lymphocytes of the blood sample by a sodium diatrizoate and polysucrose density gradient technique to obtain a lymphocytic sample;
 - (ii) centrifuging the lymphocytic sample;
 - (iii) separating and combining the lymphocytic layers from the centrifuged lymphocytic sample; and
 - (iv) washing the combined lymphocytic layers to obtain the isolated mixed T and B lymphocytes;
- (d) propagating the isolated mixed T and B lymphocytes to obtain propagated lymphocytes by:
 - (i) culturing the isolated mixed T and B lymphocytes with a cell growth medium at about 37°C;
 - (ii) centrifuging the cultured lymphocytes;

- (ii) removing the supernate from the centrifuged lymphocytes; and
- (iv) washing the centrifuged lymphocytes in normal saline with further centrifugation to obtain the propagated lymphocytes;
- (e) lysing the propagated lymphocytes to obtain a lysate by:
 - (i) suspending the propagated lymphocytes in normal saline solution;
 - (ii) sonicating the suspended lymphocytes; and
 - (iii) filtering the sonicated lymphocytes to obtain the lysate; and
 - (f) administering the lysate to the individual by:
 - (i) determining a therapeutic dose of the lysate by skin testing; and
 - (ii) injecting the individual subcutaneously with the therapeutic dose of the lysate.

61. (Previously Presented) The method according to Claim 60, wherein the cell growth medium is supplemented with bovine calf serum.

62. (Previously Presented) The method according to Claim 60, wherein the culture is monitored until the yield is approximately $5-8 \times 10^6$ cells per ml.

63. (Previously Amended) The method according to Claim 60, wherein the step of administering the lysate to the individual further comprises the step of: injecting the individual subcutaneously with at least one additional therapeutic dose of the lysate.

64. (Previously Presented) The method according to Claim 60, further comprising the steps of: measuring the clinical symptoms and signs of the individual before administering the lysate, and then measuring clinical symptoms and signs of the individual after administering the lysate.

65-66. Canceled.

67. (Previously Presented) A method according to Claim 49, wherein the step of determining the initial status of the cell cycle comprises the steps of: adding lysing buffer to a portion of the cell sample; adding DNA stain and RNase to the portion of the cell sample; analyzing the portion of the DNA stained cell sample with flow cytometry to determine the DNA distribution in the cell cycle.

68–69. Canceled.

70. (Previously Presented) A method for treating a chemically sensitive individual, the method comprising the steps of:

- (a) collecting a blood sample from the individual;
- (b) determining an initial status of the cell cycle for T lymphocytes;
- (c) isolating mixed T and B lymphocytes from the blood sample;
- (d) propagating the isolated mixed T and B lymphocytes to obtain propagated lymphocytes;
- (e) lysing the propagated lymphocytes to obtain a lysate; and
- (f) administering the lysate to the individual.

IX. EVIDENCE APPENDIX

Appellant submits as Exhibit A the 9/20/2010 Declaration of William J. Rea submitted in response to the 3/18/2010 non-final Office Action. For purposes of this appeal, Exhibit A has been renumbered to reflect pp. 26 – 82.

EXHIBIT A

[submitted on 9/20/2010 in response to
the 3/18/2010 non-final Office Action]

Application No.: 08/902,692
Attorney Docket No. 16715CIP

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application No. : 08/902,692
Filing Date : 07/30/1997
Named Inventor(s) : William J. Rea; Bertie B. Griffiths
Filed : 07/30/1997
Attorney Docket No. : 16715CIP

DECLARATION OF WILLIAM J. REA

1. My name is William J. Rea. My office address is 8345 Walnut Hill Lane, Suite 220, Dallas, Texas 75231.

2. I am over 21 years of age, of sound mind, and competent to make this Declaration. All the statements made in this Declaration made on personal knowledge are true, or, if made on information and belief, are believed to be true.

3. I graduated from Ohio State University College of Medicine in Columbus, Ohio. I then completed a rotating internship at Parkland Memorial Hospital in Dallas, Texas. I held a general surgery residency from 1963–67 and a cardiovascular surgery fellowship and residency from 1967–69 with The University of Texas-Southwestern Medical School system.

4. I am a licensed physician in the States of Texas, Ohio, Arkansas, and Illinois.

5. Among other practice certifications, I have held a practice certification from the American Board of Environmental Medicine since August 20, 1988.

6. I am the author of four medical textbooks on the subject of chemical sensitivity:

William J. Rea, Chemical Sensitivity, Vol. 1, *Principles and Mechanisms*, Lewis Pubs. (CRC Press), 1992;

William J. Rea, Chemical Sensitivity, Vol. 2, *Sources of Total Body Load*, Lewis Pubs. (CRC Press), 1994;

William J. Rea, Chemical Sensitivity, Vol. 3, *Clinical Manifestation of Pollutant Overload*, Lewis Pubs. (CRC Press), 1996; and

William J. Rea, Chemical Sensitivity, Vol. 4, *Tools of Diagnosis and Methods of Treatment*, Lewis Pubs. (CRC Press), 1997.

7. My medical textbooks on the subject of chemical sensitivity have been used in at least the following U.S. medical schools: Duke University; Brody School of Medicine at East

Carolina University; University of Texas at Ft. Worth; University of Oklahoma; and University of Kansas.

8. I have published more than 100 research papers related to the topic of thoracic and cardiovascular surgery and environmental medicine.

9. Among other offices held, I have been a Member of the Science Advisory Board for the United States Environmental Protection Agency.

10. A true and correct copy of my curriculum vitae and list of publications is attached as Exhibit A (33 pages) to this Declaration.

11. I am a named co-inventor on the above-referenced application for patent.

I. Examiner Raises Irrelevant and Unfounded Allegations

12. In the Office Action dated March 18, 2010, on page 4, the Examiner states:

... Barrett (2007) discloses a complaint filed against Inventor Rea filed with the Texas Medical Board which questions the validity of the diagnosis and treatment of chemical sensitivity as proffered by Inventor Rea. ...

13. I believe the referenced complaint is completely irrelevant and prejudicial and should not be considered.

14. On August 27, 2010, the complaint In the Matter of the License of William J. Rea, M.D., was finally resolved by Mediated Agreed Order (a settlement agreement), a true and correct copy of which is attached as Exhibit B (8 pages) to this Declaration.

15. In the Mediated Agreed Order, the Board Charges were explained as follows:

Board Staff filed a complaint at the State Office of Administrative Hearings ("SOAH") charging Respondent with violations related to five patients. The charges concerned Respondent's diagnosis and treatment of "chemical sensitivity." *After the completion of discovery, it appears that notwithstanding the allegations of the complaint, the primary concern of the Board relates to and focuses on Respondent's use of chemical antigens and the informed consent for such treatment.*

Exhibit B, In the Matter of the License of William J. Rea, Mediated Agreed Order, August 27, 2010, page 1 (*emphasis added.*)

16. The "use of chemical antigens" is not the subject matter of the above-referenced application for patent. The use of chemical antigens is not the treatment with autogenous lymphocytic factor ("ALF") as described and claimed in the application for patent.

17. This matter was the result of an anonymous third-party complaint made to the Texas Medical Board against me in 2005. This type of complaint is made to the board without the knowledge or consent of the patient involved. All five patients cited in the complaint had no knowledge that they or their information was being used in this way. Further, none of the patients ever alleged mistreatment or malpractice against me, and all five remained under my care after this complaint. Additionally, all five of these patients wrote to the Texas Medical Board and informed them that they are not part of this complaint and they are not making any allegations against me of any kind.

18. The Mediated Agreed Order did not make any factual findings against the use of the chemical antigen injections, but only states:

3. Board staff *asserts* Respondent's treatment is unsupported by medical research and is non-therapeutic. In addition, Board Staff *asserts* there was a lack of proper informed consent for these treatments.

4. Respondent asserts that his diagnosis, care, and treatment of the above patients was appropriate and in accordance with established principles of medicine and peer reviewed articles disclosed to the Board.

Exhibit B, In the Matter of the License of William J. Rea, Mediated Agreed Order, August 27, 2010, page 3, (*emphasis added*.)

19. Except regarding the Informed Consent documents, the *assertions* against the use of the antigen injections were not agreed to and not adjudicated. I vigorously disputed these assertions and maintain that they were unfounded.

20. To settle this matter, I agreed to change the Informed Consent documents used in my medical practice. Exhibit B, In the Matter of the License of William J. Rea, Mediated Agreed Order, August 27, 2010, pages 3–6.

21. In the Office Action dated March 18, 2010, on page 3–4, the Examiner states:

... In addition, regarding Inventor Rea and the use of the factor recited in the claims (aka ALF aka autogenous lymphocytic factor), Hall indicates that is unclear if ALF can actually be used to treat disease (see pages 3-4). ...

28

22. I believe the reference to Hall (2009) is completely irrelevant and prejudicial and should not be considered.

23. Hall (2009) does not address the claimed subject matter in the above-referenced application for patent.

24. Hall (2009) references a television "profile" of me on "Nightline" in 2008. Hall (2009), page 2. "Nightline" is a popular television news show, not any kind of a scientific forum.

25. In my opinion, Hall (2009) makes an unfounded personal attack against me and smears fact, and such personal attacks should not be given any evidentiary weight regarding any application for patent.

II. "Level of Skill" Regarding the Subject Matter of the Claimed Invention is High

26. In my opinion, the level of the skill regarding the subject matter of the claimed invention is high. More particularly, a person of ordinary skill in the art has the credentials of an M.D. and, in addition, is a practitioner in the specialty of environmental medicine.

III. Chemical Sensitivity Refers to Symptoms, Not to Syndrome or Other Diseases

The Chemical Environment

27. As I published in my book in 1992:

The rapidly accelerating rate of growth of modern technology has been accompanied by a proliferation of a wide variety of new toxic chemicals such as styrene, polyesters, polyethylene, etc. Recent studies¹⁻³ show that nearly 50% of the global pollutants isolated from natural products or synthesized which enter the atmosphere are generated by man. The pervasiveness of toxic chemical agents is well documented. In 1987, American industry poured 22 billion lb of toxic chemicals into the air, food, and water. Overall, Texas, ranking first in air and land releases,⁴ dumped the most pollutants. Every day several million gallons of chemicals are emptied into Lake Erie, which is the source of drinking and bathing water for most cities from

Toledo to Cleveland, OH, to Buffalo, NY. Inorganic pollutants include ozone, carbon monoxide, nitrous oxides, sulfur dioxides, heavy metals,⁵⁻¹⁰ and other metals (e.g., Al, Cu, etc.).^{11, 12} Organic pollutants include pesticides, formaldehyde,¹³ solvents (e.g., toluene and xylene), drugs,¹⁴ terpenes, cleaning chemicals, cigarette smoke, combustion products, consumer products (e.g., clothing, building materials, hygiene products, etc.),¹⁵⁻¹⁷ and biological compounds (mold toxins).^{18, 19} The most toxic organic pollutants are those classified as halogenated aromatic and aliphatic hydrocarbons.²⁰ According to the EPA,²¹ more than four million chemical compounds are currently recognized. Over 60,000 of these are produced commercially, and about 3 new compounds are introduced each day. The rampant widespread presence of hazardous chemicals in our environment has become critical.

William J. Rea, Chemical Sensitivity, Vol. 1, *Principles and Mechanisms*, Lewis Pubs. (CRC Press), 1992, pages 7–8, citations not included.

“Chemical Sensitivity” or “Chemically Sensitive” Individual Refers to Symptoms

28. “Chemical sensitivity” refers to symptoms. “Symptoms” refers to subjectively perceived problems or complaints reported by a patient. For example, a rash is a symptom that the immune system is reacting to something. “Signs” is the clinical term for symptoms, especially when observed by a physician.

29. “Chemical sensitivity” is defined as “an adverse reaction to ambient levels of chemicals generally accepted as subtoxic in our environment in air, food, and water.” William J. Rea, Chemical Sensitivity, Vol. 1, *Principles and Mechanisms*, Lewis Pubs. (CRC Press), 1992, Glossary, page 482.

30. “Chemical sensitivity” is properly established by chemical challenge testing. “Challenge tests” are “tests designed to incite a reaction in the body by any route, i.e., oral, skin, inhalation.” William J. Rea, Chemical Sensitivity, Vol. 1, *Principles and Mechanisms*, Lewis Pubs. (CRC Press), 1992, Glossary, page 482. This reaction in the body is observed by *symptoms and signs, and additionally confirmed by abnormal laboratory data*.

31. A “chemically sensitive” individual (or patient) is a person has “chemical sensitivity” or is “chemically sensitive.”

The Specification Defines and Uses “Chemically Sensitive” Referring to Symptoms

32. In the Office Action dated March 18, 2010, on page 3, the Examiner states (*emphasis added*):

... the term “chemically sensitive individual” is not specifically defined in the specification. However, based on page 12, last paragraph, and page 15, last paragraph of the specification said patients apparently encompass those suffering from dermatitis, vasculitis, asthma, organic brain syndrome, gulf war syndrome or immune system specific suppression, dysfunction or deregulation and arthritis. However, Orme et al. indicates that it is unclear if a diagnostic entity such as the “chemically sensitive individual” (*aka multiple chemical sensitivity*) with the aforementioned diseases actually occurs (see page 6, third paragraph from the bottom). ...

33. The specification of the above-referenced application for patent defines how the term “chemically sensitive” is being used in the specification and elaims:

... However, the emphasis of this invention is on the treatment of the individuals who have compromised immune systems that result in an abnormal susceptibility to environmental chemicals (*chemically sensitive*), pollens, dust, molds, food (allergies), bacteria and non-HIV viruses with reurrent infections.

Specification, page 4, lines 10– 14.

34. In addition, the specification identifies this principal characteristic of “chemically sensitive individuals”:

The total of 290 chemically sensitive individuals that were investigated in these studies were affected principally by environmental incitants found in categories such as food, biological inhalants, and chemicals.

Specification, page 13, lines 16 –18.

35. The term “chemically sensitive” individual is defined and used in the specification and claims with reference to *symptoms, signs, and abnormal laboratory data*.

The Application and Claims Are *Not* Directed to “Multiple Chemical Sensitivity”

36. The above-referenced application for patent does *not* use the term “multiple chemical sensitivity.” The claims are *not* directed to “multiple chemical sensitivity.”

“Multiple Chemical Sensitivity” (“MCS”) Refers to a Syndrome

37. “Multiple chemical sensitivity” refers to a *syndrome*, not to symptoms, *signs*, and *abnormal laboratory data*.

38. A medical definition of “*syndrome*” is the association of several clinically recognizable features, symptoms, phenomena, or characteristics that often occur together, so that the presence of one feature alerts to the presence of the others. The term *syndrome* derives from Greek, and it literally means “run together.” At least initially, the word refers to a set of detectable characteristics that run together when the pathophysiology has not yet been discovered.

39. In 1987, Mark C. Cullen proposed the syndrome of “multiple chemical sensitivity” (abbreviated as “MCS”) as having seven diagnostic criteria: (1) some documentable environmental exposures, insults, or illness at onset; (2) symptoms affect more than one organ system; (3) symptoms recur and subside in response to predictable stimuli; (4) symptoms occur when exposed to different chemicals and toxins; (5) symptoms are caused by proven exposures; (6) exposures that produce symptoms must be very low (far below average levels); and (7) no common test of organ-system function can explain symptoms. Cullen, M. R., The worker with multiple chemical hypersensitivities: An overview, State of the Art Review, *Occupational Medicine* 2, 655-661. According to Cullen (1987), the criteria should not be restrictive and describe a sizeable patient population, as there are many patients who meet “some but not all of [the criteria]” (Cullen, p. 658).

40. In my opinion, such a “multiple chemical sensitivity” syndrome has not been established. Nevertheless, people do suffer from chemical sensitivity, as shown by *symptoms*, *signs*, and *abnormal laboratory data*, and as demonstrable by chemical challenge testing.

“Chemical Sensitivity” Symptoms Are Not Multiple Chemical Sensitivity” Syndrome

41. The terms “chemical sensitivity” and “chemically sensitive” individual should be understood as defined and used in the specification and as would be understood by a person of

skill in the field to which the application particularly pertains. These terms should *not be equated with the syndrome of "multiple chemical sensitivity."*

The Claims Are Not Directed to Other Diseases

42. In the Office Action dated March 18, 2010, on page 3, the Examiner stated (*emphasis added*):

... the term "chemically sensitive individual" is not specifically defined in the specification. However, based on page 12, last paragraph, and page 15, last paragraph of the specification said patients *apparently* encompass those suffering from dermatitis, vasculitis, asthma, organic brain syndrome, gulf war syndrome or immune system specific suppression, dysfunction, or deregulation and arthritis.

43. As explained above, "chemically sensitive" individual is defined in the specification, and does not refer to "multiple chemical sensitivity" syndrome.

44. Regarding other diseases, additional relevant text of the cited paragraphs from the specification is provided below:

Clinical Testing and Results

Twenty-five (25) individuals were used as normal controls. A total of 290 individuals were tested, including a first test group of 100 patients, and a second test group of 190 patients. The vast majority of these individuals were chemically sensitive, chronically ill patients, including those suffering from dermatitis, vasculitis, asthma, organic brain syndrome, gulf war syndrome, or immune system suppression, dysfunction, or deregulation. In addition, three (3) of the patients that were tested suffered from cancer, and one (1) was HIV positive.

About five percent (5%) of the individuals that otherwise would have been in the studies could not tolerate ALF. Except for noting this fact, these patients were not included in the data because they did not take enough ALF to be evaluated.

...

The total of 290 chemically sensitive individuals that were investigated in these studies were affected principally by environmental incitants found in categories such as food, biological inhalants, and chemicals. They presented" histories of varied backgrounds, but common among them was that all showed

irregular cell cycles including T and B lymphocytes and subset numbers and functions. ...

Specification, page 12, line 17 – page 13, line 20.

45. This cannot be construed as the examiner “apparently” suggests. In the full context of the application, this statement is properly interpreted as referring to the 290 individuals as having *at least* the common denominator of being chemically sensitive, not that “chemical sensitivity ... apparently encompasses” the listed the symptoms and diseases.

46. In my opinion and experience, chemically sensitive individuals, as a class, frequently *additionally* suffer from one or more of a wide variety of other diseases. Nevertheless, other diseases are not part of the definition of “chemical sensitivity.” Regarding “chemical sensitivity,” the question of a causal connection to other diseases is irrelevant. “Chemical sensitivity” is not necessarily the cause of various other diseases. Only “chemical sensitivity” symptoms are required.

IV. Evidence of Treating An Irregular Cell Cycle for T Lymphocytes

The Examiner Overstates the Scope of the Claimed Invention

47. In the Office Action dated March 18, 2010, on page 4, the Examiner states (*emphasis added*):

Regarding the use of the claimed method to regulate an abnormal lymphocytic cell cycle of continuously dividing *T and B lymphocytes* in a mammal wherein said method encompasses the in vivo treatment of humans, there is no evidence in the specification that such regulation has been achieved using the elaimed method. Regarding Wands factors 1-3, the claimed method encompasses a method wherein according to the specification the abnormal lymphocytic cell cycle of continuously dividing *T and B lymphocytes in a mammal is normalized*.

48. Pending claim 49 is directed to “A method for treating a chemically sensitive individual having an irregular cell cycle for *T lymphocytes*” Claim 70 is similar to pending Claim 49 *except* that the preamble does *not* include the language “having an irregular cell cycle for T lymphocytes.”

49. In addition, the specification and claims do not require the irregular cell cycle for T lymphocytes be “normalized.” Of course, this is a desired goal, but the claims are directed to “treating,” which is illuminated by the specification:

... treating the individual with a therapeutic amount of the ALF, and determining the individual's lymphocytic cell cycle to observe any regulatory effect on the lymphocytic cell cycle and subsets.

Specification, page 6, lines 16–18.

... As treatment [with ALF] continued, in general, in about six weeks a more drastic shift toward that of a normal profile was observed.

Specification, page 14, lines 8–9 (*emphasis added*).

The specification discloses: “The autogenous lymphocytic factor (ALF) appears to act as a modulator since total lymphocytes, and T4 and T8 lymphocytes in particular, significantly elevated or decreased in order to obtain normalization.” Specification, page 17, lines 4–6. In addition:

According to the presently most preferred embodiment of the invention, the method can be used to establish a basis for the regulation of an individual's T lymphocytes that are observed to be irregular due to varied incitants; thus restoring normal T lymphocyte functions and the ability of a compromised individual to cope with multiple insults to his/her immune system.

Specification, page 14, lines 15–21, as amended on April 23, 2008 (*emphasis added*). This language does not require complete regulation, but is a basis for regulation of the cell cycle.

The Normal Cell Cycle for Both T and B Lymphocytes Was Well Known

50. In the Office Action dated March 18, 2010, on page 4–5, the Examiner states:

The only actual data disclosed in the specification wherein the cell cycle of human cells is analyzed is that represented in Figures 2–4. Figures 2a and 2b represent data indicating the cell cycle of human peripheral T lymphocytes from “normal” volunteers. This data provides no information about the cell cycle of human peripheral B lymphocytes from “normal” volunteers.

51. However, the "normal" cell cycle was well known in the art at the time the invention was made and the application was filed:

FIG. 1 is a *diagrammatic representation of a normal mammalian cell cycle*, wherein the overall cell doubling time is about 20 - 24 hours, the G₁ phase lasting about 8 - 12 hours, the S phase lasting about 6 - 8 hours, the G₂ phase lasting about 3 - 5 hours, and the M phase lasting about 0.5 - 1 hour.

Specification, page 5, lines 13-16 (*emphasis added*).

52. It would be understood by a person of skill that this description of the cell cycle in Figure 1 refers generally to the cell cycle applicable to both T and B lymphocytes, which are continuously-dividing cells, also known as labile cells. Labile cells are cells that multiply constantly throughout life. For example, it is well known that if the cell cycle for such labile cells is normal, the cells should spend little or no time in the quiescent G₀ phase of the cell cycle, but rather should regularly perform cell division.

53. In addition, it was well known in the art at the time the invention was made and the application was filed that the cell cycles for T and B lymphocytes are substantially similar.

54. It would be understood by a person of skill in the art that Figure 1 is an idealized representation of the cell cycle, and that there is individual variation in the actually measured cell cycles.

55. Further, the specification states: "FIG. 2a is a normal DNA histogram of human peripheral T lymphocytes. FIG. 2b is a representative cell cycle DNA histogram obtained from "normal" or "control" volunteers." Specification, page 8, lines 1-2.

56. The specification discloses: "Furthermore, *it is possible to independently establish as a matter of trivial routine experiment the norms for the lymphocytic cell cycle.*" Specification, page 8, lines 13-15 (*emphasis added*).

The Specification Evidences Improvement in the Cell Cycle for T Lymphocytes

57. In the Office Action dated March 18, 2010, on page 5, the Examiner continues (*emphasis added*):

... *The specification indicates that abnormal lymphocytic cell cycle of continuously dividing T and B lymphocytes in a mammal is regulated. Figures 3a-3e purport to show the "irregular cell cycle*

profiles from environmentally compromised individuals". There is no disclosure as to what cells are referred to in said figure (e.g., only T cells, T and B cells, unfractionated lymphocytes, unfractionated leukocytes, etc.). Thus, it is unclear if there is any relationship between the data disclosed in Figure 2 and that in Figure 3 because it is unclear whether said Figures refer to the same or different cell populations. A similar problem exists with the data represented in Figure 4. Furthermore, if the data disclosed in Figure 4 refers to the cell cycle of T cells, it appears that the cell cycle of untreated patients in Figure 4a more closely approximates that seen in the normal controls than that seen in Figure 4c. Thus, the evidence of record suggests that the claimed method cannot be used to "regulate" the cell cycle of abnormal lymphocytic cell cycle of continuously dividing T and B lymphocytes because the term regulate would encompass normalization of an abnormal cell cycle and this has not been demonstrated. In addition, based on the data presented in the specification, it is unclear whether any effect on the cell cycle of continuously dividing B lymphocytes in a mammal has been achieved. ...

58. As stated above, pending claim 49 is directed to: "A method for treating a chemically sensitive individual having an irregular cell cycle for *T lymphocytes*"

59. The specification also states: "The cell cycle presents a *reflection of the status of the T lymphocytes* in an individual." Specification, Page 14, line 15.

60. In addition, the specification teaches:

To determine the lymphocyte cell cycle, and the T and B lymphocytes and subset counts of the individual, heparanized lymphocytes can be used;

Cells tagged fluorometrically for DNA content are analyzed in a flow cytometer ... and such equipment and techniques are well known to those skilled in the art. This information provides a 'snapshot' of the individual's present cell cycle. T and B cells and subsets counts are preferably also measured on the flow cytometer.

Specification, page 7, line 15–23.

61. It is clear from the specification that unless otherwise specified, the "cell cycle" refers to the cell cycle for mixed T and B lymphocytes, including all subsets, which is disclosed to present a reflection of the status of the T lymphocytes, including all subsets. No other interpretation is reasonable.

62. In addition, it would be known to a person of skill in the art at the time the invention was made and the application was filed that T cells are generally well over 10 times more numerous than B cells. The specification is consistent with this. *See, e.g.*, Specification, Tables 7 and 17. Thus, a DNA histogram of combined T cells and B cells would typically be a reflection primarily of the status of the peripheral T lymphocytes. The specification was written to a person of skill in the art, who would understand this.

63. Accordingly, the “cell cycle” histogram for mixed T and B lymphocytes is reflective of the well-known normal cell cycle for T lymphocytes.” Indeed, the “cell cycle” for mixed T and B lymphocytes includes the cell cycle for T lymphocytes, and is predominately that of T lymphocytes. This is clear.

64. Whether the cell cycle is determined specifically for T lymphocytes or it is based on mixed T and B lymphocytes, the cell cycle it is a reflection of the status of T lymphocytes.

65. Among other evidence provided in the specification, the written description states:

Significant changes were typically observed in patients treated with ALF. Changes were observed in improvement of overall clinical manifestations and immune studies. With regard to clinical manifestations, minimal symptoms (which were improved over the onset symptoms) continued after three weeks of continued therapy with ALF. Immunologically, there were significant regulations of lymphocytic cell cycles, especially from one phase of the cycle to another, and changes in T and B lymphocyte cell numbers and functions. Patients became less sensitive to exposures and more tolerant to specific irritants. As treatment continued, in general, in about six weeks a more drastic shift toward that of a normal profile was observed.

Specification, page 14, lines 1–8 (*emphasis added*).

66. The patients who could not take ALF did not exhibit such dramatic improvement.

67. Figures 4a–c represents a *single case history*. These figures illustrate to a person of skill in the art a drastic improvement in the cell cycle, which is a reflection of the improvement of the cell cycle for T lymphocytes. The progression of these figures shows a shift from a cell cycle in which the majority of cells were predominately “stuck” in the G₂–M phase. This is an abnormal cell cycle compared to the normal cell cycles illustrated in Figures 1 and 2a

and 2b. Figure 4c shows that the cell cycle for the patient had been drastically shifted after about six weeks toward the normal cell cycle, in which the majority of cells are in the G₀-G₁ phase.

The Claims Do Not Require Regulation in Patients Suffering from Autoimmune Disease

68. In the Office Action dated March 18, 2010, on page 5, the Examiner states:

There is no evidence provided that the claimed invention can be used to regulate the abnormal lymphocytic cell cycle of continuously dividing T and B lymphocytes in patients suffering from autoimmune disease.

69. The specification states:

It is anticipated that the invention can also be applied to the study of the prevention and/or treatment of some cancers. Being a biological response modifier, and having proven efficacies in certain non-HI V viral infections, the invention is also expected to stimulate the immune system of immuno-compromised individuals, thus, it is expected that HIV-positive individuals might be benefitted. The invention is expected to be useful in the study of the dysfunctional and suppressed immune system of HI V-positive individuals, which may also result in a therapy.

Specification, page 4, lines 14–20.

70. The application is not claiming to have had proof of regulating a cell cycle that may be abnormal for any conceivable reason.

The Results Obtained are Too Large to Attribute to Other Treatments

71. In the Office Action dated March 18, 2010, on page 5, the Examiner states:

Furthermore, the treated patients also received other treatments (for example see page 15) so it is unclear as to what treatments contributed to the "results" obtained in the specification in the absence of an appropriate control group.

72. In my experience as a practitioner, the results obtained with the treatment were much faster, much more positive, and much higher in occurrence among the 290 chemically sensitive patients described than could be attributed to the placebo effect or any of the other therapies or treatments that had been used until that time in environmental medicine. In my

opinion, a person of skill in the field would appreciate the improvements indicated by the data presented, including in the tables of the specification, as being unprecedented and highly indicative that ALF was responsible for such a large effect. This evidence would be generally recognized by those of ordinary skill in the art as convincing evidence of the asserted utility of the described and claimed methods.

A Theoretical Explanation Was Offered in the Specification

73. In the Office Action dated March 18, 2010, on page 5, the Examiner states:

It is also noted that the number of cells used in the procedure disclosed in pages 9-10 to prepare ALF would yield a protein preparation with a concentration of any particular protein that would be far below that used for any biological modifier used to treat humans. For example, the use of rituximab in humans requires a dosage of approximately 750 mg per patient wherein said quantity requires billions to cells to produce such a quantity of molecule.

74. Without limiting the invention to any particular theoretical explanation, the specification offered a preliminary theoretical discussion of the preliminary clinical successes as reported in the application. Specification, page 22, line 20 – page 23, line 17.

V. No Undue Experimentation Required

75. Preliminary evidence was provided at the time of filing the application of some highly successful treatments according to the claimed invention, in rate of response, degree of response, and frequency of response that could only be attributed to the new treatment with ALF.

76. The specification provides a “cook book” example of the procedure for practicing the invention.

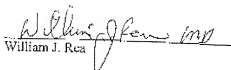
77. The statements contained in the written description regarding the scope of the claimed invention as set forth in the presently pending claims are supported by the data presented.

78. As discussed above, the level of the skill regarding the subject matter of the claimed invention is high.

79. Based on the description in the specification and the above factors, there is no requirement for "undue experimentation." A person of skill in the art, based on the invention disclosure and with good financial and time resources, could conduct additional testing and clinical trials using the invention to elucidate the cause-and-effect relationships involved.

VI. Declaration

80. I hereby declare that all statements made herein of my own knowledge are true, that all statements made on information and belief are believed to be true, and further that these statements are made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under § 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application, and patent issuing thereon, or any patent to which this verified statement is directed.


William J. Rea

9-20-2010
Date

WILLIAM J. REA, M.D.

**Bus: 8345 Walnut Hill Lane
Suite 220
Dallas, Texas 75231-4262
214/368-4132**

PERSONAL: Born: Jefferson, Ohio
February 2, 1935

COLLEGES: Otterbein College
Westerville, Ohio
Graduated with B.S. and B.A., 1958

Ohio State University College of Medicine
Columbus, Ohio
Graduated with M.D., 1962

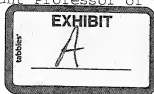
INTERNSHIP: Parkland Memorial Hospital
Dallas, Texas
Rotating Internship: 1962 - 1963

RESIDENCIES: University of Texas Southwestern Medical School
- General Surgery Residency 1963-1967
Parkland Memorial Hospital
Baylor Medical Center
Veteran's Hospital
Children's Medical Center
University of Texas Southwestern Medical School
- Cardiovascular Surgery Fellowship and Residency
1967-1969

LICENSURE: License #D2294, Dallas County, Texas
License #35-02-5922-R, Franklin County, Ohio
License #R-3120, Poinsett County, Arkansas
License #036-085797, Fangamon County, Illinois

**TEACHING
APPOINTMENTS:** First World Professorial Chair, Environmental
Medicine, Robens Institute, University of
Surrey, Guildford, Surrey, England, 1988 - 1995
Adjunct Associate Professor of Environmental
Sciences and Mathematics at the University of
Texas, 1984 - 1985
Chief of Surgery, Brookhaven Hospital, Dallas,
Texas, 1980 - 1981
Clinical Associate Professor of Thoracic Surgery,
University of Texas Southwestern Medical School,
1972 - 1982

TEACHING 42 Assistant Professor of Thoracic Surgery,



William J. Rea, M.D.

APPOINTMENTS:

University of Texas Southwestern Medical
School 1969 - 1972
Chief of Thoracic Surgery, Veteran's Hospital
1969 - 1972
Guest lecturer, University of Texas at Dallas 1985 -
1988, Adjunct Professor of Environmental Sciences
Guest lecturer, North Texas State University, 1980,
1982 - 1988 - Adjunct Professor of Psychology
Adjunct Professor, Department of Occupational
and Environmental Health, The University of
Oklahoma, 1992 - 1993
Regional Clinical Faculty, Kirksville College of
Osteopathic Medicine, 1994 - 1996
Consultant, Institut für Umweltkrankheiten, Bad
Emstal, Emstal, Germany
Regional Clinical Faculty, Kirksville College of
Osteopathic Medicine, 1995 - 1997
Professor of Medicine, Capital University of
Integrative Medicine, Washington, D.C., July 1,
1997 - 2005

PRACTICE:

Private Practice - June 1, 1973 to present

CERTIFICATION:

American Board of Surgery Certification
April 1, 1968, Certificate #15416
American Board of Thoracic Surgery
April 4, 1970, Certificate #2137
American Board of Environmental Medicine
August 20, 1988, Certificate #003
American Board Certification, Disability Analyst,
#5513-01

MEDICAL SOCIETY

MEMBERSHIPS:

Past/Present

American Medical Association
Texas Medical Association
Dallas County Medical Society
American Heart Association (Golden Heart
Member)
American Lung Association
American Society of Artificial Internal Organs
Pan American Medical Association
Society of Thoracic Surgeons
Correspondence Society of Surgeons
American Board of Surgery
American Board of Thoracic and
Cardiovascular Surgery

William J. Rea, M.D.

MEDICAL SOCIETY

MEMBERSHIPS:

Past/Present

Association for Academic Surgery
American Academy of Environmental Medicine
Pan American Allergy Society
American Occupational Medical Association
American Association for Clinical Immunology
and Allergy
American Institute of Medical Climatology
Huxley Institute for Biosocial Research
American College of Nutrition
American College of Surgeons
Chirurgio Society
The New York Academy of Sciences
The American College of Preventive Medicine
Oklahoma College of Occupational Medicine
(Section on Environmental Medicine)
American Board of Forensic Examiners
Society of Integrative Medicine
International Society for Heart and Lung
Transplantation
The International Society for the Study of
Subtle Energies and Energy Medicine
American College of Occupational and
Environmental Medicine

FELLOWSHIPS:

Fellow, American College of Surgeons
Fellow, American Academy of Environmental
Medicine
Fellow, American College of Preventive
Medicine
Fellow, American College of Nutrition
Fellow, Royal Society of Medicine
(Cardiothoracic Section)
Honorary Fellow, International Academy of
Preventive Medicine
Affiliate Fellow, American Academy of
Otolaryngic Allergy
Distinguished Fellow, American Academy of
Cardiology

MEMBERSHIPS/

OFFICES HELD:

Past/Present

Director, Environmental Health Center -
Dallas
President, American Environmental Health
Foundation
Past President, Board of Directors, Member,
Pan American Allergy Society 1988-1989

William J. Rea, M.D.

**MEMBERSHIPS/
OFFICES HELD:
Past /Present**

Vice President of the Board, American
Board of Environmental Medicine
Chief of Surgery, Brookhaven Medical Center
Member, Science Advisory Board, The United
States Environmental Protection Agency
Member, Research Committee, American
Academy of Otolaryngic Allergy
Member, Board of Directors and Past President,
American Academy of Environmental Medicine
Member, American Association for the
Advancement of Science
Member, The Smithsonian Society
Member, National Advisory Board, American
Security Council
Member, National Geographic Society
Member, Technology Committee, American
College of Allergists
Member, Food Allergy Committee, American
College of Allergists
Member, Editorial Board, Clinical
Ecology, Archives for Human Ecology in
Health and Disease
Member, Committee on Aspects of
Cardiovascular, Endocrine & Autoimmune
Diseases, American College of Allergists
Member, Board of Advisers, CAMPRO - The
Center for Accelerated Medical Progress,
Inc.
Editorial Board, Journal Bioelectricity and
Electromagnetic Fields - Marino
Member, Human Ecology Action League Director,
ECHO-HAVEN
Member, World Research Foundation
Member, Chief Executive Officer's Club
Editorial Advisory Board, Medical Nutrition
National Board of Directors - Natural Food
Associates
Northwest Coalition for Alternatives to
Pesticides
Chief Patron, Ecosystem Research Foundation in
Pakistan
Member, Mycotoxin Steering Committee, World
Health Organization
Board of Scientific Directors, Brain
Allergy Research

William J. Rea, M.D.

**MEMBERSHIPS/
OFFICES HELD:
Past/Present**

Member, Human Ecology, Academy of
Technological Science of Russian Federation
Scientific Advisory Board, Biosphere II
Editorial Board, Journal of Environmental
and Waste Management
Advisory Board, Price-Pottenger Nutrition
Foundation
Advisory Board, Latitudes
Editorial Board, Management of Environmental
Quality
Honorary Member, Nutrition For Optimal Health
Association
Member, Overseas Editorial Board, Journal
of Environmental Biology
Director, Orthomolecular Health-Medicine
Editorial Board, Journal of Long-Term Effects
of Medical Implants
Advisor to Board, American Academy of
Environmental Medicine

**SPECIAL
AWARDS:**

The Jonathan Forman Gold Medal Award by
the American Academy of Environmental
Medicine - 10/31/87
Mountain Valley Water Hall of Fame, Hot
Springs, Arkansas 1987
The Special Achievement Award by
Otterbein College - 06/15/91
The Herbert J. Rinkel Award by the
American Academy of Environmental
Medicine, 10/11/93
The Distinguished Pioneers in Alternative
Medicine Award by the Foundation for the
Advancement of Innovative Medicine Education
Fund - 5/1/94
Gold Star Award by the International
Biographical Center, December 1997
Five Hundred Leaders of Influence, 1997
Who's Who In the South and Southwest, 1997
The Twentieth Century Award For Achievement, 1997
American Board of Environmental Medicine Service
Award 1998

**SPECIAL
AWARDS:**

Dor W. Brown, Jr., M.D. Lectureship Award, Pan
American Allergy Society, March 2002
O. Spurgeon English Humanitarian Award, Temple
University, October 2002
Professor Emeritus of Integrative Medicine, Capital
University of Integrative Medicine, June 2006.

William J. Rea, M.D.

PUBLICATIONS

1. Rea, W.J. and Robertson, W.O.: Serum Salicylate Levels (their importance in predicting toxicity). Ohio Medical Journal 59: July, 1963.
2. Rea, W.J., Graview, L. and Jennings, R.L.: Liver Angioma of Neonate. American Journal of Surgery 112: pp. 777; November, 1966.
3. Sugg, W.L., Rea, W.J., Ecker, R.R., Rose, E., Webb, W.R. and Shaw, R.R.: Penetrating Wounds of the Heart: An Analysis of 459 Cases. Journal of Thoracic Cardiovascular Surgery 56: pp. 531-545; October, 1968.
4. Rea, W.J., Sugg, W.L., Wilson, L.C., Webb, W.R. and Ecker, R.R.: Coronary Artery Lacerations (An analysis of 22 patients). Annals of Thoracic Surgery 7: pp. 518-528; June, 1969.
5. Chawla, Narinder, P.S., Rea, W.J. and Shapiro, W.: The Use of an Implanted Demand Pacemaker in Bradyarrhythmias. Archives of Internal Medicine 124: pp. 593-599; November, 1969.
6. Sugg, W.L., Rea, W.J., Webb, W.R. and Ecker, R.R.: Cardiac Assistance (Counterpulsations) in Ten Patients: Clinical and Hemodynamic Observations. Annals of Thoracic Surgery 9: pp. 1-12; January, 1970.
7. Rea, W.J., Wyrick, W. and McClelland, R.N.: Rare Complications of Intravenous Hyperosmolar Alimentation. JAMA 211, 10: March 9, 1970.
8. Rea, W.J., Wyrick, W., McClelland, R.N. and Webb, W.R.: Intravenous Hyperosmolar Alimentation. Geriatrics Digest 7: September 28, 1970 (Abstract).
9. Rea, W.J. and Wyrick, W.: Necrotizing Fasciitis. Annals of Surgery 172: pp. 957-964; December, 1970.
10. Rea, W.J., Wyrick, W. and McClelland, R.N.: Intravenous Hyperosmolar Alimentation. Archives of Surgery 100: pp. 393-398; 1970.
11. Ecker, R.R., Libertini, R.V., Rea, W.J., Sugg, W.L., Webb, W.R.: Injuries of the Trachea and Bronchi. Annals of Thoracic Surgery 11: pp. 289-298; April, 1971.

William J. Rea, M.D.

12. Rea, W.J., Wyrick, W.: Necrotizing Fasciitis. Modern Medicine, 39 No. 17: pp. 77-78; August 23, 1971.
13. Ecker, R.R., Mullins, C.B., Grammar, J.C., Rea, W.J., Atkins, J.M.: Control of Intractable Ventricular Tachycardia by Coronary Revascularization. Circulation 44 Vol. 4: pp. 666-670; October, 1971.
14. Rea, W.J., Eberle, J.W., Watson, J.T., Ecker, R.R., Sugg, W.L.: Gas Transfer in a Heparinized Membrane Oxygenator. Surgical Forum Vol. XXII: pp. 188-190; October, 1971.
15. Rea, W.J., Ecker, R.R., Sugg, W.L.: Zinc in Cardiac Surgery. Journal of Surgical Residents 13, No. 3: pp. 164-167; March, 1972.
16. Ecker, R.R., Rea, W.J., Sugg, W.L., Miller, W.W.: Changes in 2, 3-Diphosphoglycerate after Cardiopulmonary Bypass. Annals of Thoracic Surgery 13: pp. 364-370; April, 1972.
17. Rea, W.J., Mills, C., Whitley, D., Eberle, J.W., Sugg, W.L.: Assessment of Long Term Membrane Oxygenation Without Heparin. European Society for Experimental Surgery, Abstracts, p. 288, 7th Congress Amsterdam, The Netherlands, April 11-14, 1972.
18. Schwade, J., Pombo, J., Rea, W.J., Waksman, A., Shapiro, W.: Emergency Diagnosis and Attempted Therapy in Post-Infarction Interventricular Septal Rupture. Texas Medicine Vol. 68: pp. 74-78; May, 1972.
19. Rea, W.J., Gallivan, G.J., Ecker, R.R., Sugg, W.L.: Traumatic Esophageal Perforation. Annals of Thoracic Surgery 14: pp. 671-677; December, 1972.
20. Rea, W.J., Eberle, J.W., Ecker, R.R., Watson, J., Sugg, W.L.: Long-Term Membrane Oxygenation in Respiratory Failure. Annals of Thoracic Surgery, 1972.
21. Rea, W.J.: Hyperalimentation in Chest Surgery. In Intravenous Hyperalimentation. Philadelphia, Lea & Febiger, Chapter 15, pp. 177-184; 1972.
22. Rea, W.J., Whitley, D., Eberle, J.W.: Long-Term Membrane Oxygenation Without Systemic Heparinization. Society of Artificial Internal Organs, 1972.

William J. Rea, M.D.

23. Whitley, D.E., Meals, C.R., Eberle, J.W., Manton, J.R., Somerville, P., Rea, W.J.: 24-Hour Membrane Oxygenation Without Systemic Heparin. Surgical Forum ACS, 1972.
24. Rea, W.J.: Myocardial Revascularization. Proceedings of John Peter Smith Hospital, Tarrant County Hospital District, Spring, 1972. Vol. 3: pp. 52-53.
25. Rea, W.J.: Ecologic Aspects of Cardiovascular Disease. International Academy of Preventive Medicine, 1975.
26. Rea, W.J.: Environmentally Triggered Thrombophlebitis. Annals of Allergy, Vol. 37, pp.101-109; July, 1976.
27. Rea, W.J., Dickey, L.C.: Relationship of Food and Chemical Sensitivities to Acute Respiratory Failure, Chapter 14; and Ecologic Orientation and Thoracic and Cardiovascular Surgery, Chapter 73. Clinical Ecology, Charles C. Thomas, Publisher, 1976.
28. Rea, W.J., Ecologic Orientation in Thoracic and Cardiovascular Surgery, Clinical Ecology, ed. Lawrence D. Dickey, Publisher Charles C. Thomas; Springfield, Illinois, 1976.
29. Rea, W.J.: Environmentally Triggered Small Vessel Vasculitis. Annals of Allergy, Vol. 38: pp. 245-251; April, 1977.
30. Rea, W.J.: Environmentally Triggered Cardiac Disease. Annals of Allergy, Vol. 40: pp. 243-251; April, 1978.
31. Rea, W.J., Bell, I.R., Suits, C.W., Smiley, R.E.: Food and Chemical Susceptibility after Environmental Chemical Overexposure: Case Histories. Annals of Allergy, August, 1978.
32. Edgar, R.T., Fenyes, E.J., Rea, W.J.: Air Pollution Analysis Used in Operating and Environmental Control Unit. Annals of Allergy, March, 1979.
33. Rea, W.J., Johnson, F. (editor): Parotiditis Sicca: Management of Environmentally Triggered Allergic Disease in Allergy: Including IgE in Diagnosis and Treatment. Symposia Specialists, 1979.
34. Rea, W.J.: The Environmental Aspects of Ear, Nose and Throat Disease: Part I. J.C.E.O.R.L. & Allergy, July, 1979.

William J. Rea, M.D.

35. Rea, W.J.: The Environmental Aspects of Ear, Nose and Throat Disease: Part II. J.C.E.O.R.L. & Allergy, September, 1979.
36. Rea, W.J.: Diagnosing Food and Chemical Susceptibility. Continuing Education: pp.47-59; September, 1979.
37. Rea, W.J., Edgar, R.T., Fenyves, E.J., Smiley, R.E., Stroud, R.M.: Comprehensive Air Pollution Measurements In An Environmental Unit, Part I. Annals of Allergy, Vol. 42, No. 3, March 1979.
38. Rea, W.J., Bell, Iris, R. and Smiley, R.E., Johnson, F. and Spencer, J.T. (Eds.). Allergy: Immunology and Medical Treatment, Symposia Specialists, 1980.
39. Rea, W.J., Suits, C.W., Gerrard, John W., D.M. (Ed.): Cardiovascular Disease Triggered by Foods and Chemicals. Food Allergy: New Perspectives: pp.99-143; Publisher Charles C. Thomas, 1980.
40. Rea, W.J.: Review of Cardiovascular Disease in Allergy. Bi-Annual Review of Allergy: pp. 282-347; Claude Frazier (Ed.), 1979-80.
41. Rea, W.J., Peters, D.W., Smiley, R.E., Edgar, R., Greenberg, M., Fenyves, E.J.: Recurrent Environmentally Triggered Thrombophlebitis (A 5-Year Follow Up). Annals of Allergy, December, 1981.
42. Rea, W.J.: Environmental Aspects of Ear, Nose and Throat Disease. Otolaryngologic Allergy, H.C. King, M.D. (Ed.), Symposia Specialists, Inc., Miami, Florida, 1981.
43. Rea, W.J.: Environmental Allergy; Sandorama, 1981.
44. Rea, W.J.: Fibroscopic Bronchoscopy. Bi-Annual Review of Allergy, Claude A. Frazier, M.D. (Ed.), 1981-82.
45. Rea, W.J.: Review of Cardiovascular Disease in Allergy. Bi-Annual Review of Allergy, Claude A. Frazier, M.D. (Ed.), 1981-82.
46. Rea, W.J.: Environmentally Triggered Disorders. Sandorama, IV: pp. 27-31; 1982.
47. Rea, W.J., Mitchell, M.J.: Chemical Sensitivity and The Environment, Immunology & Allergy Practice, September/ October 1982, pp. 157/21-167/31.

William J. Rea, M.D.

48. Rea, W.J.: Review of Cardiovascular Disease in Allergy. Bi-Annual Review of Allergy, Claude A. Frazier, M.D. (Ed.), 1982-83.
49. Rea, W.J.: Intermittent Unilateral Leg Swelling. Correspondence Society of Surgeons, Vol. 6, No. 3, March, 1983.
50. Rea, W.J.: The Environmental Aspects of Cardiovascular Disease. Spectrum, Vol. XXIV, No. 2, International, pp. 25-30, 1983.
51. Rea, W.J.: Review of Cardiovascular Disease in Allergy. Bi-Annual Review of Allergy, Claude A. Frazier, M.D. (Ed.), 1984-85.
52. Rea, W.J.: Elimination of Oral Food Challenge Reaction by Injection of Food Extracts: A Double-Blind Evaluation. Archives of Otolaryngology, American Medical Association, April, 1984, Vol. 110, pp. 248-252.
53. Laseter, J.L., Ildefonso, R.D., Rea, W.J., Butler, J.R.: Chlorinated Hydrocarbon Pesticides in Environmentally Sensitive Patients. Clinical Ecology, Vol. II, No. 1, pp. 3-12; 1983.
54. Laseter, J.L., Ildefonso, R.D., Rea, W.J., Butler, J.R.: Pesticides and Brain-Function Changes In A Controlled Environment. Clinical Ecology, Vol. II, No. 3, pp. 145-150; 1984.
55. Marshall, R.T., Stroud, R.M., Kroker, G.F., Bullock, T., Carroll, F.M., Greenberg, M., Randolph, T.G., Rea, W.J., Smiley, R.: Fasting and Rheumatoid Arthritis: A Multicenter Study. Clinical Ecology, Vol. II, No. 3, pp. 137-144; 1984.
56. Marshall, R.T., Stroud, R.M., Kroker, G.F., Bullock, T., Carroll, F.M., Greenberg, M., Randolph, T.G., Rea, W.J., Smiley, R.: Food Challenge Effects On Fasted Rheumatoid Arthritis Patients: A Multicenter Study. Clinical Ecology, Vol. II, No. 4, pp. 181-190; 1984.
57. Bertschler, J., Butler, J.R., Lawlis, G.F., Rea, W.J., Johnson, A.R.: Psychological Components of Environmental Illness: Factor Analysis of Changes During Treatment. Clinical Ecology, Vol. III, No. 2, pp. 85-96; 1985.

William J. Rea, M.D.

58. Rea, W.J., Brown, O.D.: Mechanisms Of Environmental Vascular Triggering. Clinical Ecology, Vol. III, No. 3, pp. 122-128, 1985.
59. Rea, W.J., Brown, O.D.: Clinical Aspects Of Sensitivity To Chemicals: Use Of An Environmental Unit, In Freed, D.L.J. (Ed.), Controversial Techniques In Allergy, England, 1985.
60. Rea, W.J., Johnson, A.R., Smiley, R.E., Maynard, B., Dawkins-Brown, O.: Magnesium Deficiency In Patients With Chemical Sensitivity. Clinical Ecology, Vol. IV, No. 1, pp. 17-20; 1986.
61. Rea, W.J., Johnson, A.R., Youdim, S., Fenyves, E.J., Samadi, N.: T And B Lymphocyte Parameters Measured in Chemically Sensitive Patients And Controls. Clinical Ecology, Vol. IV, No. 1, pp. 11-14; 1986.
62. Rea, W.J.: Correspondence Society of Surgeons, 1986, Vol. 6, No. 2, pp. 4-5 Response to Steven M. Lib, M.D., N. Miami Beach, FL., Dye induced pancreatitis.
63. Rea, W.J., Dawkins-Brown, O.: Environmental Aspects of Cardiovascular Disease. Brostoff, J., and Challacombe, S.J. (Eds.), London: Bailliere Tindall, Food Allergy & Intolerance, pp. 737-753, 1987.
64. Rea, W.J.: Inter-relationships Between The Environment And Premenstrual Syndrome. M.G. Brush and E.M. Goudsmit (Eds.), John Wiley & Sons Ltd. Functional Disorders of the Menstrual Cycle.
65. Rea, W.J., Pan, Yaquin, Laseter, J.L., Johnson, A.R., Fenyves, E.J.: Toxic Volatile Organic Hydrocarbons In Chemically Sensitive Patients, Clinical Ecology, Vol. V, No. 2, 1987.
66. Rea, W.J.: The Environmental Effects on Neurovascular Problems, Neuro-ophthalmology Japan (Japanese Journal of Neuro-ophthalmology), Vol. 4, No. 4, 1987.
67. Rea, William J., Pan, Yaquin, Johnson, Alfred R., Fenyves, Ervin J.: T & B Lymphocytes in Chemically Sensitive Patients With Toxic Volatile Organic Hydrocarbons in Their Blood, Clinical Ecology, Vol. V, No.4, pp.171-175, 1987/88.

William J. Rea, M.D.

68. Rea, William J., Pan, Yagin, Johnson, Alfred R.: Clearing of Toxic Volatile Hydrocarbons From Humans, Clinical Ecology, Vol. V, No. 4, pp. 166-170, 1987/88.
69. Pan, Yagin, Johnson, Alfred R., Rea, William J.: Aliphatic Hydrocarbon Solvents in Chemically Sensitive Patients, Clinical Ecology, Vol. V, No. 3, pp. 126-131, 1987/88.
70. Rea, W.J.: Chemical Hypersensitivity and the Allergic Response, Ear, Nose and Throat Journal, Volume 67, January, 1988.
71. Rousseau, David, Rea, W.J., M.D., Enwright, Jean: Your Home, Your Health, and Well-Being, Hartley and Marks, Vancouver, B.C., 1988.
72. Rea, W.J., Ross, G.H.: Food and Chemicals as Environmental Incitants, The Nurse Practitioner: The American Journal of Primary Health Care, Sept. 1989.
73. Ross, G.H., Rea, W.J., Johnson, A.R., Maynard, B.J., Carlisle, L.: Evidence for Vitamin Deficiencies in Environmentally-Sensitive Patients, Clinical Ecology, Vol. VI, No. 2, pp. 60-66, 1989.
74. Rea, William J., Ross, Gerald H., Johnson, Alfred R., Smiley, Ralph E., Fenyves, Ervin J.: Chemical Sensitivity in Physicians, Clinical Ecology, Vol. VI, No. 4, pp. 135-141, 1989.
75. Rea, William J., Ross, Gerald H., Johnson, Alfred R., Smiley, Ralph E., Sprague, Donald E., Fenyves, Ervin J., Samadi, N.: Confirmation of Chemical Sensitivity by Means of Double-Blind Inhalant Challenge of Toxic Volatile Chemicals, Clinical Ecology, Vol. VI, No. 3, 1989.
76. Khan, A., Rea, W.J., Johnson, A., Ahmed, Shafi: Possible Use of Co-Enzyme Q-10 in L-Tryptophan Associated Eosinophilia-Myalgia Syndrome, 1990, in press.
77. Rea, William J.: Response to Collected Letters in Correspondence Society of Surgeons, April 1990.
78. Pan, Y., Rea, W.J., Johnson, A.R., Fenyves, E.J.: Formaldehyde Sensitivity, Clinical Ecology, Volume VI, No. 3, 1990.
79. Youdim, S., Rea, William, Liang, H.: Treatment of

William J. Rea, M.D.

- Environmentally Sensitive Patients With Transfer Factor, Part I: Immunologic Studies, Clinical Ecology, Volume 7, No. 3, pp. 55-61, 1990.
80. Youdim, S., Rea, William: Treatment of Environmentally Sensitive Patients With Transfer Factor, Part II: Clinical Studies and Immunological Correlates, Clinical Ecology, Volume 3, pp. 62-66, 1990.
81. Youdim, S., Liang, H., Rea, William: Treatment of Environmentally Sensitive Patients With Transfer Factor, Part III: Case Studies on Three Patients, Clinical Ecology, Volume 3, pp. 67-72, 1990.
82. Rea, William J.: Environmental Pollutants (Editorial), Asociación Médica de Puerto Rico, Volume 83, No. 7, pp. 278-279, July 1991.
83. Rea, William J., Ross, Gerald H.: Food and Chemicals as Environmental Incitants, Asociación Médica de Puerto Rico, Volume 83, No. 7, pp. 310-315, July 1991.
84. Pan, Y., Johnson, Alfred R., Rea, William J.: Aliphatic Hydrocarbon Solvents in Chemically Sensitive Patients, Asociación Médica de Puerto Rico, Volume 83, No. 7, pp. 316-320, July 1991.
85. Shirakawa, S., Ishikawa, S., Tsujisawa, I., Rea, W.J.: Disturbance of the Autonomic Nervous System in the Presence of Organochlorine Pesticide -- Pupillographic Study, Journal of Ophthalmologica, in press.
86. Rea, William J., Liang, Hsueh-Chia: Effects of Pesticides on the Immune System, Journal of Nutritional Medicine, Volume 2, pp. 399-410, 1991.
87. Rea, William J., Pan, Yaqin, Fenyes, Ervin J., Sujisawa, Iehiko, Nasrola, Samadi, Ross, Gerald H.: Electromagnetic Field Sensitivity, Journal of Bioelectricity, Volume 10, No. 1 & 2, 1991.
88. Kuehn, Kevin A., Johnson, Alfred R., Rea, William J.: Ascomycetes: A Potential Aeroallergen?, Submitted to Proceedings of the Texas Academy of Science 94th Annual Meeting, Stephen F. Austin State University, 1991.

William J. Rea, M.D.

89. Kuehn, Kevin A., Johnson, Alfred R., Rea, William J.: Preliminary Observation on the Airborne Mycofloral Component Within The North Dallas, Texas Metropolitan Area. Environmental Medicine, Volume 8, No. 3, pp. 79-91, 1991.
90. Shirakawa, S, Rea, William J., Ishikawa, S., Johnson, A.: Evaluation of the Autonomic Nervous System Response By Pupillographical Study in the Chemically Sensitive Patient. Environmental Medicine, Volume 8, No. 4, pp. 121-127, 1991.
91. Rea, William J., Johnson, Alfred R., Ross, Gerald H., Butler, Joel R., Fenyves, Ervin J., Griffiths, Bertie, Laseter, John: Considerations for the Diagnosis of Chemical Sensitivity. (Addendum to Biologic Markers in Immunotoxicology) National Academy Press, p.169-192, 1992.
92. Rea, William J.: Chemical Sensitivity, Volume I, Lewis Publishers, Boca Raton, Florida, 1992.
93. Kuehn, Kevin A., Garrison, Robert, Robertson, Larry, Koehn, Robert D., Johnson, Alfred R., Rea, William J.: Identification of Airborne Microfungal Populations from Home Environments within the Dallas-Fort Worth (Texas) Region, Indoor Environment, Volume 1, No. 5, 1992, pp. 285-292, Karger AG, Basel, Switzerland.
94. Liang, Hsueh-chia , Rea, William J., Fenyves, Ervin J.: Study on the Critical Reference Value From the Regression Equations Between Chlorinated Pesticides and Immune Parameters. Environmental Medicine, Volume 9, No. 1, pp. 10-16, 1992.
95. Livingston, Linda, Sanjar, Mansour R., Rea, William J.: Toluene Sensitivity As Part of the Chemical Sensitivity Syndrome - Case Report. Environmental Medicine, Volume 9, No. 2, pp. 60-72, 1992.
96. Simon TR, Hickey DC, Rea WJ, Johnson AR, Ross GH, Fincher C, Harrell EH and Kettlehut M. Breast implants and organic solvent exposure can be associated with abnormal cerebral SPECT studies in clinically impaired patients. Radiological Society of North America, 1992. Radiology 185(P):234, 1992.
97. Rea, William J., et al: Considerations for the Diagnosis of Chemical Sensitivity. Talmage, I.N., et al. Biological Matters in Immunotherapy, Washington, D.C., National Academy Press 1992 p. 169

William J. Rea, M.D.

98. Rea, William J.: Biometeorology, Part 2, Volume 1: Proceedings of The 13th International Congress of Biometeorology (Under the Auspices of The International Society of Biometeorology), Calgary, Alberta, Canada, September, 1993, pp. 26-53.
99. Rea, William J.: The Environmental Aspects of Chemical Sensitivity, Japanese Journal of Clinical Ecology, Vol. 3, No. 1, 1994.
100. Rea, William J.: Chemical Sensitivity, Volume 2, Lewis Publishers, Boca Raton, Florida, 1994.
101. Wang, T., Hawkins, L.H., Rea, William J.: Effects of ELF Magnetic Fields on Patients With Chemical Sensitivities, GRAZ conference. 1994.
102. Higuchi, H., Miyata, M., Ishikawa, S., Rea, William J.: (1996) Abnormalities of the Autonomic Nervous System Seen In Patients Who Have Silicon Breast Implants. International Journal of Occupational Medicine, Immunology, and Toxicology, Vol. 5, No. 1. pp. 1-13.
103. Rea, William J.: Correspondence Society of Surgeons, Collected Letters, Vol. 17, No. 5, May 1994.
104. Rea, William J.: Klinische Okologie, Angewandte Umweltmedizin, Hippokrates Verlag GmbH, Stuttgart, Germany December 1994.
105. Simon, T.R., Hickey, D.C., Rea, W.J., Johnson, A.R., Ross, G.H., Fincher, C.E., and Kettlehut, M. (1992): Breast implants and organic solvent exposure can be associated with abnormal cerebral SPECT studies in clinically impaired patients. Radiological Society of North America. Radiology 185(P):234.
106. Rea, William J. (1995) Chapter 2: Why Children are More Susceptible to Environmental Hazards. In The Healthy School Handbook, ed. Miller NL. pp. 39-49, Washington, DC: NEA Professional Library.
107. Rea, William J.: Pesticides - Review. Journal of Nutritional & Environmental Medicine, Issue 5:4, December 1995.
108. Rea, William J.: Progress on Persian Gulf War Illnesses-Reality and Hypotheses. International Journal of Occupational Medicine and Toxicology, Vol. 4, No. 3, 1995.

William J. Rea, M.D.

109. Rea, William J.: Chemical Sensitivity, Volume 3, Lewis Publishers, Boca Raton, Florida, 1996.
110. Rea, William J.: Chemical Sensitivity, Volume 4, Lewis Publishers, Boca Raton, Florida, 1997.
111. Rea, William J., Pan, Y.: Fat and Blood Levels of Toxic Chemicals in Chemically Sensitive Patients. Journal of Nutritional and Environmental Medicine, United Kingdom, London, issue 5, 387-390, March 1996.
112. Rea, W. J., Pan, Y., Johnson A.R., Ross, G.H., Suyama, H., Fenyves, E.J.: Reduction of Chemical Sensitivity By Means of Heat Depuration, Physical Therapy, and Nutritional Supplementation in a Controlled Environment. Journal of Nutritional and Environmental Medicine, United Kingdom, London. 1996.
113. Rea, W.J.: Environmental Medicine and Individual Health. Ecology of Health. Sage Publications, Thousand Oaks, California, 1996.
114. Rea, W.J.: Pesticides, Journal of Nutritional & Environmental Medicine (1996) 6, 55-124.
115. Griffiths, B., Rea, W.J., Johnson A.R., Ross, G.H.: Mitogenic Effects of Mycotoxins on T4 Lymphocytes. The Faculty Press, Cambridge, Great Britain. Microbios 86, 127-134 1996.
116. Fincher, C.E, Chang, Te-Shang, Harrell, E.H., Kettelhut, M.C., Rea, W.J., Johnson, A.R., Hickey, D.C. and Simon, T.R.: Comparison of Single Photon Emission Computed Tomography Findings in Cases of Healthy Adults and Solvent-Exposed Adults, American Journal of Industrial Medicine 31:4-14 (1997).
117. Fox, R., Ross, G.H., Johnson, A., Rea, W.J.: Building Related Illness: A Patient With Sensitization To An Anti-corrosive Agent Used In Steam Humidification.
118. Rea, William J.: Correspondence Society of Surgeons, Collected Letters. Vol. 29, No. 2, February 1997.
119. Bascon, R., Meggs, W.J., Frampton, M., Hudnell, K., Kilburn, K., Kobal, K., Medinsky, M., and Rea, W.: Neurogenic Inflammation: With Additional Discussion of Central and Perceptual Integration of Nonneurogenic Inflammation.

William J. Rea, M.D.

- Environmental Health Perspectives. Vol. 105, Supplement 2, March 1997.
120. Smith, C.W., Rea, W.J., Griffiths, B.: The Frohlich Approach Approach to Cellular Communication Systems. Effects of Electricity & Magnetism in the Natural World, Section 10: Work on Cell Cultures at the Environmental Health Center, Dallas" - Madeira 1-6 Oct. 1998.
121. Baird, Deborah N., M.D., Rea, William J., M.D.: The Temporomandibular Joint Implant Controversy: A Review of Autogenous/Alloplastic Materials and Their Complications. Journal of Nutritional & Environmental Medicine (1998) 8, 289-300. Carfax Publishing Ltd.
122. Griffiths, Bertie B., Rea, William J., Griffiths, Bradley, Pan, Y.: The Role of the T-Lymphocytic Cell Cycle and An Autogenous Lymphocytic Factor in Clinical Medicine. Cytobios 93, 49-66, 1998.
123. Baird, Deborah N., M.D., Rea, William J., M.D.: The Temporomandibular Joint Implant Controversy: Part II: Its Clinical Implications. Journal of Nutritional & Environmental Medicine (1999) 9, 209-222. Taylor & Francis Ltd.
124. Ross, Gerald H., Rea, William J., Johnson, Alfred R., Hickey, David C., Simon, Theodore R.: Neurotoxicity in single photon emission computed tomography brain scans of patients reporting chemical sensitivities. Toxicology and Industrial Health, (1999), 15, 415-420.
125. Rea, William J., M.D. and Pan, Yaqin, M.D.: Toxic encephalopathy. Environmental Management and Health, Volume 11, No. 3, pp. 250-262, 2000.
126. Rea, William J., M.D., Fenyves, Ervin J., Ph.D., Seba, Douglas, Ph.D., Pan, Yaqin, M.D.: Organochlorine Pesticides and Chlorinated Hydrocarbon Solvents in the Blood of Chemically Sensitive Patients. Journal of Environmental Biology, Volume 22 (3), 163-169, 2001.
127. Harrell, E., DiGriksen, N., Butler, J.R. and Rea, W.J. (March 2001) Validation of the Comprehensive Neuropsychological Screen (CNS) in Environmental Patients. Society for Behavioral Medicine, Seattle, WA.

William J. Rea, M.D.

128. Callaway, T.G., Didriksen, N.A., and Rea, W.J. (September 2000) Clinical Analysis Questionnaire: Psychological Profiles of Neurotoxically-Exposed Patients. Texas-Oklahoma Joint Annual Convention, Texas and Oklahoma Psychological Associations, Dallas, TX.
129. Rea, William J. and Pan, Yaqin: The use of the environmental control unit to study indoor air pollution. Dimensions of Pollution, Vol. 1, pp. 104-114, 2002.
130. Rea, W.J. and Ross, G.H.: Cardiovascular Disease in Response to Foods and Chemicals. Food Allergy & Intolerance, Brostoff and Challacombe, 2nd Edition. Saunders 2002.
131. Rea, William J.: Optimum Environments for Optimum Health and Creativity. Crown Press 2003.
132. Simon, Theodore R. and Rea, William J.: Use of Functional Brain Imaging in the Evaluation of Exposure to Mycotoxins and Toxins Encountered in Desert/Storm/Desert Shield. Archives of Environmental Health, July 2003, Heldref Publications.
133. Rea, William J., Didriksen, Nancy, Simon, Theodore R., Pan, Yaqin, Fenyves, Ervin J., and Griffiths, Bertie: Effects of Toxic Exposure Associated With Neurobehavioral and Pulmonary Impairment: A Preliminary Report. Archives of Environmental Health, July 2003, Heldref Publications.
134. Curtis, L., Lieberman, A., Stark, M., Rea, W. and Vetter, M.: Adverse Health Effects of Indoor Molds. Journal of Nutritional and Environmental Medicine, September 2004, 14(3), 261-274.
135. Curtis, L., Rea, W., Smith-Willis, P., Fenyves, E. and Pan, Y: Adverse Health Effects of Outdoor Air Pollutants. Environment International 32, Issue 6 (2006) 815-830.
136. Curtis, L., Lieberman, A., Stark, M., Rea, W. and Vetter, M.: Adverse Health Effects of Indoor Moulds. Nexus Magazine, J June-July 2006, V. 13, No. 4, p. 19-23.
137. Dean, Amy L., Rea, William J., Curtis, Luke: Environmental Therapy-Induced Remission of Aplastic Anemia. Journal of Nutritional & Environmental Medicine, August 2007, V. 16, Issue 3 & 4, p. 227-231.

William J. Rea, M.D.

138. Rea, William J., Pan, Yaqin, Griffiths, B.: The Treatment of Patients With Mycotoxin-induced Disease, Toxicology and Industrial Health 25(9-10) 711-714.
139. Yu, George, Rea, William: Toxic Chemical Levels in Intra-Abdominal Fat Compartments Compared to Subcutaneous Fat Compartment and to Blood Serum. Environmental Health Perspectives, September 2009, IN PRESS.
140. Rea, William J., Patel, Kalpana: Reversibility of Chronic Degenerative Disease and Hypersensitivity, V. 1: Regulating Mechanisms of Chemical Sensitivity. CRC Press, June 2010.

LECTURES

Environmental Protection Agency Science Advisory Board.
Society for Clinic Ecology.
Royal Society of Medicine in London, McCarrison Society.
Charing Cross Medical School, London, England.
University of Southampton, Southampton, England.
Postgraduate Seminar in England.
Society for Clinical Ecology, Royal College of Physicians,
London, England.
Wyoming Postgraduate Course in Allergy and Immunology.
University of Texas Postgraduate Course in Allergy and
Immunology.
University of Miami Postgraduate Course in Allergy and
Immunology.
World Food Symposium, Mexico City, Mexico.
American College of Allergy.
American College of Preventive Medicine.
Tennessee State Medical Society.
World Food Symposium, Boston, Massachusetts.
Nine Wells Medical School, Dundee, Scotland.
University of Berlin.
British Society of Clinical Ecology.
Royal Australian College of Surgeons.
Straub Clinic, Honolulu, Hawaii.
London Neurological Institute, Middlesex Medical School.
First World Conference on Indoor Air Pollution, Harvard.

Indoor Air Pollution, University of Calgary School of
Architecture, Calgary, Canada.
University of Texas, School of Architecture.
American Lung Association.

William J. Rea, M.D.

Pan American Allergy Society.
The World Conference I, II, III, IV, and V on Man and His Environment
in Health and Disease.
The American Academy of Otolaryngic Allergy.
The University of Guangzhou, Guangzhou, China.
The Capital University Medical School, Beijing, China.
The 4th Military Medical School, Xian, China.
The University of Nanjing Medical School, Nanjing, China.
Wuxi Medical School, Wuxi, China.
Hang Chou Medical Society, Hang Chou, China.
Postgraduate courses for the Academy of Otolaryngic Allergy of
10 years.
The postgraduate advanced seminars for the American Academy of
Environmental Medicine for 15 years.
Postgraduate courses on chemical sensitivity for four years.
Human Ecology Action League, Washington, D.C.
German Conference on Environmental Medicine, Black Forest,
Germany
Environmental Health Association, Prince Edward Island, Canada.
World Research Foundation Conference, Los Angeles, California.
Kitasato University, Dept. of Ophthalmology, Tokyo, Japan.
Second Japanese Conference in Neurophthalmology, Matsuyama, Japan.
Environmental Medicine Foundation, Philadelphia, PA.
Emerging Challenges in Occupational and Environmental Health.
Annual New England Occupational Medical Association Conference With the
Harvard School of Public Health.
Pan American Allergy Society Postgraduate courses for physicians, San
Antonio, Texas
Pan American Allergy Society Seminar, San Antonio, Texas
The University of Rome, Rome, Italy.
The University of Puerto Rico, Mayaguez, Puerto Rico.
The British Society of Clinical Ecology, Torquay, England.
St. Hughes College, Oxford, England, McCarrison Society.
European Community Research Center, ISPRA, Lugnano, Italy.
Clinical Ecology Seminar, Eubia, Greece.
National Association for the Advancement of Science.
University of Wuhan, Wuhan, China.
Peking Union Medical School, Peking, China.
Third Military Medical School, Chong Ching, China.
American College of Nutrition.
The Ontario Postgraduate Course in Environmental Medicine,
Toronto, Canada.
The Halifax Nova Scotia Environmental Health Association.
The Minneapolis Environmental Health Association.
The California Medical Association Scientific Advising Committee.
The Province of Ontario Advisory Committee, The Effects of
Environmental Chemicals on Humans.
Southwestern Psychological Association, Austin, Texas.

William J. Rea, M.D.

German Conference, Clinical Ecology, Emstal, Germany.
Roger Wyburn-Mason & Jack M. Blount Foundation for the Eradication
of Rheumatoid Disease, Inc., Los Angeles, California.
International Polio Conference, Warm Springs, Georgia.
Natural Food Associates, Hot Springs, Arkansas.
Dalhousie University Medical School, Halifax, Nova Scotia.
Environmental Health Association, Halifax, Nova Scotia.
Texas County Medical Society, Waco, Texas.
Northeast Community Hospital, Bedford, Texas.
North Texas State University, Denton, Texas.
Sixth Annual Veterans Conference, Claremore, Oklahoma, May, 1987.
Ashrae Conference, Washington, D.C., May, 1987.
G.I.N.I.'s Fourth International Polio and Independent Living
Conference, St. Louis, Missouri, June, 1987.
The 11th International Congress of Biometeorology, West
Lafayette, Indiana, September, 1987.
American Academy of Otolaryngic Allergy, Chicago, Illinois,
September, 1987.
The 25th Japanese Congress on Neuro-ophthalmology, Japan, October,
1987.
Ninth Annual New England Occupational Health Conference, Boston,
Massachusetts, December, 1987.
American Academy of Environmental Medicine, 12th Instructional
Course, Denver, Colorado, December, 1987.
1988 Allergy: In-Vitro, Orlando, Florida, February, 1988.
Sixth Annual International Symposium on Man and His Environment in
Health and Disease, February 1988.
Pan American Allergy Society, San Antonio, Texas, March, 1988.
Australian Society for Environmental Medicine, Melbourne, Australia,
March, 1988.
Australia's Association of Chemical Victory, March, 1988.
Tasmanian Medical Society, Tasmania, Australia, March, 1988.
Health By Choice, Atlanta, Georgia, April, 1988.
The Canadian Society for Clinical Ecology and Environmental
Medicine, April, 1988.
T.V. Ontario, Ontario, Canada, May, 1988.
V.O.T.E. Environmental Awareness Symposium, Oklahoma City,
Oklahoma, June, 1988.
Rocky Mountain Environmental Health Association, Denver,
Colorado, August, 1988.
American Academy of Otolaryngic Allergy, Washington, D.C.,
September, 1988.
4th International Symposium for Environmental Medicine, Emstal,
Germany, October, 1988.
T.V. Ontario, Ontario, Canada, October, 1988.
American Academy of Environmental Medicine, 22nd Scientific Session,
Incline Village, Nevada, October, 1988.
Asahikawa Medical University, Asahikawa, Japan, November, 1988.

William J. Rea, M.D.

Kitasato University, Tokyo, Japan, November, 1988.
American Academy of Environmental Medicine, 13th Instructional
Course, Part III, Cleveland, Ohio, December, 1988.
Allergy In-Vitro Update, Phoenix, Arizona, December, 1988.
T.V. Ontario, Ontario, Canada, February, 1989.
University of Toronto, Toronto, Canada, February, 1989.
Seventh Annual Symposium on Man and His Environment in Health and
Disease, Dallas, Texas, February, 1989.
Pan American Allergy Society, San Antonio, Texas, March, 1989.
Clinical Ecology Study Group, Ft. Worth, Texas, April, 1989.
Ft. Worth Club, Ft. Worth, Texas, April, 1989.
American College of Advancement in Medicine, Dallas, Texas, May,
1989.
Natural Food Associates, Atlanta, Texas, June, 1989.
Environment Week, Moncton, New Brunswick, Canada, June, 1989.
American Academy of Environmental Medicine, Denver, Colorado,
July, 1989.
U.S. House of Representatives Committee on Science, Space, and
Technology, Washington, D.C., July, 1989. Testified.
American Academy of Otolaryngic Allergy, New Orleans, Louisiana,
September, 1989.
British Society for Nutritional Medicine, London, England,
September, 1989.
National Society for Research into Allergy, Enfield, England,
September, 1989.
American Academy of Otolaryngic Allergy, New Orleans, Louisiana,
September, 1989.
American Academy of Environmental Medicine, Atlanta, Georgia,
October, 1989.
Second National Conference on Pesticides and Human Health,
Cirencester, England, October, 1989.
American Academy of Otolaryngic Allergy, Corpus Christi, Texas,
November, 1989.
Eighth Annual International Symposium on Man and His Environment in
Health and Disease, Dallas, Texas, February, 1990.
Pan American Allergy Society, San Antonio, Texas, March, 1990.
The Environmental Medicine Foundation, London, England, April, 1990.
British Society for Allergy and Environmental Medicine, Buxton,
Derbyshire, England, July 1990.
American Academy of Environmental Medicine, Fifteenth Instructional
Course, Minneapolis, Minnesota, July, 1990.
American Academy of Otolaryngic Allergy Annual Meeting, San Diego,
California, September, 1990.
American Society of Otolaryngic Allergy Technicians, San Diego,
California, September, 1990.
Workshop to Review Congressional Office of Technology Assessment's
Document on Identifying and Controlling Immunotoxic Substances,

William J. Rea, M.D.

September, 1990.
Building Pathology 90, Surrey, England, September, 1990.
Pesticides Conference, Breakspear Hospital, Hertfordshire, England,
September, 1990.
Fifth International Symposium on Environmental Medicine, Emstal,
Germany, September, 1990.
The American Academy of Environmental Medicine 25th Annual Meeting,
Coeur d'Alene, Idaho, October, 1990.
Oklahoma College of Occupational Medicine 15th Annual Fall
Educational Meeting, Edmond, Oklahoma, November, 1990.
American Academy of Otolaryngic Allergy, Newport Beach, California,
February, 1991.
Ninth Annual International Symposium on Man and His Environment in
Health and Disease, Dallas, Texas, February, 1991.
Pan American Allergy Society, San Antonio, Texas, March, 1991.
National Academy of Sciences, Irvine, California, March, 1991.
Pesticide Exposure Group of Sufferers, Cambridge, England, April, 1991.
Allergy, Nutrition and Health Preservation, Orlando, Florida,
April, 1991.
American College of Occupational Medicine, San Francisco,
California, April, 1991.
First International Symposium on "Prophylactic Role of Clean
Environment in Health Preservation", Cracow, Poland, June 1991.
First Annual Conference of the International Society for the Study
of Subtle Energies and Energy Medicine, Golden, Colorado, June
1991.
American Academy of Environmental Medicine, Sixteenth Instructional
Course, Shamburg, Illinois, July 1991.
St. John's Regional Medical Center, Joplin, Missouri, August 1991.
The 21st Century Medicine Conference, Czechoslovakia, August 1991.
American Academy of Otolaryngic Allergy, Kansas City, Missouri,
September 1991.
American Academy of Environmental Medicine, Jacksonville, Florida,
October 1991.
American College of Occupational Medicine, St. Louis, Missouri,
October 1991.
American Academy of Otolaryngic Allergy, Orlando, Florida, November
1991.
Tenth Annual International Symposium on Man and His Environment in
Health and Disease, Dallas, Texas, February 1992.
Pan American Allergy Society, Houston, Texas, March 1992.
The University of Oklahoma Health Sciences Center, College of
Public Health, Oklahoma City, Oklahoma, April 1992.
Klaire Europe Nutrition '92 Seminar, Amsterdam, May 1992.
American College of Advancement in Medicine, Dallas, Texas, May
1992.
Allergy Problems in Buildings, London, England, June 1992.
American Academy of Otolaryngic Allergy, Washington, D.C.,

William J. Rea, M.D.

September 1992.
Fourth Annual James R. Miller Conference on Brain Function and Learning, University of North Texas, September 1992.
Seventh Symposium fur Umweltmedizin, Emstal, Germany, September, 1992.
American Academy of Environmental Medicine, Lincolnshire, Illinois, October 1992.
American Academy of Otolaryngic Allergy, Las Vegas, Nevada, October 1992.
Oklahoma College of Occupational and Environmental Medicine, Fall Occupational Health Conference, Norman, Oklahoma, November 1992.
Tenth Annual International Symposium on Man and His Environment in Health and Disease, Dallas, Texas, February 1993.
Pan American Allergy Society, Houston, Texas, March 1993.
Health Built in the Environment, Calgary, Alberta, March 1993.
American College of Occupational and Environmental Medicine, Atlanta, Georgia, April 1993, **completion of ACOEM Curriculum in Occupational Medicine.**
American Academy of Environmental Medicine, Schaumburg, Illinois, April 1993.
Second International Conference on Nanometer Scale Science and Technology, Moscow, Russia, August 1993.
Diagnosztikus es Terapeutikus Modszerek, Budapest, Hungary, August 1993.
International Congress of Clinical Ecology, Asahikawa, Japan, September 1993.
13th International Congress of Biometeorology, Calgary, Canada, September 1993.
VIII Symposium fur Umweltmedizin, Bad Emstal, Germany, September 1993.
American Academy of Otolaryngic Allergy, Minneapolis, Minnesota, September/October 1993.
American Academy of Environmental Medicine, Reno, Nevada, October 1993.
American Academy of Pain Management, Annual Conference, Knoxville, Tennessee, October 1993.
American College of Occupational and Environmental Medicine, Dallas, Texas, October 1993.
American Academy of Otolaryngic Allergy, Key Biscayne, Florida, November 1993.
Twelfth International Symposium on Man and His Environment in Health and Disease, Dallas, Texas, February 1994.
Pan American Allergy Society, Houston, Texas, March 1994.
Environmental Allergy Update, University of Osteopathic Medicine and Health Sciences, Des Moines, Iowa, April 1994.
American Academy of Environmental Medicine, Kansas City, Missouri, April 1994.
Foundation for the Advancement of Innovative Medicine Education Fund, Inc., New York, New York, April 1994.
Symposium on Developing a Research Strategy for Investigating Multiple Chemical Sensitivity", California Department of

William J. Rea, M.D.

Health Services, Environmental Health Investigations Branch, B
Berkeley, California, May 1994.
Primer Simposio Latino Americano De Salud Ambiental, Rosario,
Argentina, May 1994.
Fourth International Symposium, Food and Environmental Factors in Human
Disease, London, England, June 1994.
Occupational Rhinitis Symposium, York, England, June 1994.
Examining Research Assumptions in Alternative Medical Systems,
National Institutes of Health, Bethesda, Maryland, July 1994.
Fourth International Symposium and Workshops on Inner Ear Medicine
and Surgery, Snowmass-Aspen, Colorado, July 1994.
Clinical Ecology Study Group, Fort Worth, Texas, August 1994.
American Academy of Otolaryngic Allergy, San Diego, California,
September 1994.
Fourth International Scientific Conference, Work With Display Units,
Milan, Italy, October 1994.
American Academy of Environmental Medicine Twenty-Ninth, Virginia
Beach, Virginia, October 1994.
Rocky Mountain Environmental Health Association, Denver, Colorado,
November 1994.
Multiple Chemical Sensitivity, A Seminar for the Naturopathic
Academy of Allergy and Environmental Medicine, Bellevue,
Washington, November 1994.
American Academy of Otolaryngic Allergy, Phoenix, Arizona, November
1994.
Thirteenth Annual International Symposium on Man and His
Environment in Health and Disease, Dallas, Texas, February
1995.
Society for Orthomolecular Medicine America, San Francisco,
California, March 1995.
Pan American Allergy Society, San Antonio, Texas, March 1995.
American Academy of Environmental Medicine, Phoenix, Arizona, March
1995.
Earth Week, Experimental Approaches to Chemical Sensitivities,
Evanston, Illinois, April 1995.
American Academy of Environmental Medicine, Houston, Texas, May 1995.
2nd Copenhagen Conference on Electromagnetic Hypersensitivity,
Copenhagen, Denmark, May 1995.
American Academy of Otolaryngic Allergy, New Orleans, Louisiana,
September 1995.
10th International Symposium for Environmental Diseases, Bad
Emstal, Germany, September 1995.
American Academy of Environmental Medicine 30th Annual Meeting,
Tucson, Arizona, September 1995.
Australian Conference of Environmental Medicine, Brisbane, Australia,
November 1995.
Environmental Health and Cardiovascular Disease, Morristown, NJ,
December 1995.

William J. Rea, M.D.

- Fourteenth International Symposium, on Man and His Environment in Health and Disease, Dallas, Texas, February 1996.
- American Academy of Environmental Medicine Spring Board Meeting, Cancun, Mexico, March 1996.
- Pan American Allergy Society, Training Course and Seminar, San Antonio, Texas, March 1996.
- American Academy of Environmental Medicine, Part III, Dearborn, MI, April 1996.
- American Academy for Advanced Medicine, Orlando, Florida, May 1996.
- 4th International Symposium, Nutritional, Orthomolecular and Minimally Invasive Anterior Surgery of the Lumbar Spine, Memphis, Tennessee, June 14-15, 1996.
- Environmental Modalities in Medical Practice, Salzburg, Austria, July 1996.
- Australian Conference of Environmental Medicine, Toxicity '96, Understanding, assessing and managing diseases caused by exposure to toxins. Brisbane, Australia, August 31 - Sept 1. 1996.
- American Academy of Otolaryngic Allergy, Annual Meeting Scientific Program. Washington, D.C., September 26-28, 1996.
- American Academy of Environmental Medicine, 31st Annual Meeting, Boston, MA, October 11-15, 1996.
- American College for Advancement in Medicine, Palm Springs, California, October 31 to November 3, 1996.
- The American College of Allergy, Asthma & Immunology, Boston, MA, November 8-13, 1996.
- Fifteenth International Symposium, on Man and His Environment In Health and Disease, Dallas, Texas February 1997.
- Pan American Allergy Society, Annual Training Course and Seminar, San Antonio, TX. March 19-23, 1997.
- American Academy of Environmental Medicine, Spring Instructional Courses, Kansas City, MO. April 17-22, 1997.
- Sociedad Mexicana De Alergia en Otorrinolaringología, Guadalajara, Mexico, January 24, 1998.
- American Academy of Environmental Medicine, Instructional Courses, Potomac, MD. April 2-7, 1998.
- American Academy of Otolaryngic Allergy, 57th Annual Meeting, San Antonio, TX. September 10-12, 1998.
- American Academy of Environmental Medicine, 33rd Annual Meeting, Baltimore, MD. November 6-8, 1998.
- Seventeenth Annual International Symposium on Man and His Environment in Health and Disease, Dallas, TX. June 10-13, 1999.
- The Health Impact of Chemical Exposures During the Gulf War: A Research Planning Conference, Atlanta, GA. February 28- March 2, 1999.
- Pan American Allergy Society, 43rd Annual Training Course and

William J. Rea, M.D.

- Seminar, San Antonio, TX. March 10-14, 1999.
- American Academy of Environmental Medicine, Instructional Courses, St. Charles, IL. March 21-26, 1999.
- "A Healthy Home and School for Your Child", Richardson Church of the Nazarene, Richardson, TX. April 20, 1999.
- American Academy of Otolaryngic Allergy, New Orleans, LA. September 23-25, 1999.
- American Academy of Environmental Medicine, Coeur d'Alene, ID. October 10-12, 1999.
- 4th International Congress of Bioenergetic Medicine, Orlando, Fl. February 25-27, 2000.
- Pan American Allergy Society, San Antonio, TX. March 8-12, 2000.
- 12th International Symposium, Integrative Medicine, Lisbon, Portugal. Portugal, June 22-25, 2000.
- National CPA Health Care Advisors Association, HCAA-Sponsored Health Care Track, Chicago, Illinois, July 19-21, 2000.
- Anti-Aging Conference & Exposition, Chicago, Illinois, July 22-23, 2000.
- Fifty-Ninth Annual Meeting, American Academy of Otolaryngic Allergy, Washington, D.C., September 21-23, 2000.
- American Academy of Environmental Medicine, Hilton Head, SC, September 27-30, 2000.
- Seminars on Scientific Aspects of Fluoridation, San Antonio, TX, October 14, 2000.
- Endometriosis Association 20th Anniversary Conference, Milwaukee, WI, October 21, 2000.
- American College for Advancement in Medicine, Advanced Anti-Aging Workshop, Salt Lake City, UT, October 25-26, 2000.
- American Academy of Otolaryngic Allergy, Austin, TX, November 30 - December 3, 2000.
- International Symposium on Current Status of Indoor Air Pollution by Organic Compounds and Countermeasures for Healthy Housing, Tokyo Japan, January 13, 2001.
- La Sociedad Mexicana de Alergia en Otorrinolaringología, Guadalajara, Mexico, February 1-3, 2001.
- Pan American Allergy Society, San Antonio, Texas, March 7-11, 2001.
- American Academy of Environmental Medicine, Colorado Springs, Colorado, March 29-31, 2001.
- 19th International Symposium on Man & His Environment in Health & Disease, Dallas, Texas, June 7-10, 2001.
- American Academy of Otolaryngic Allergy, Denver, Colorado, September 6-8, 2001.
- Texas Conference Medical-Dental Outreach Congress, Corpus Christi, Texas, October 4-6, 2001.
- New England Conference on Health, Environment, and Medicine, Farmington, Connecticut, October 13, 2001.
- American Academy of Environmental Medicine, Colorado Springs, Colorado, October 18-20, 2001.

William J. Rea, M.D.

- Texans for Alternatives to Pesticides, Dallas, Texas, March 5, 2002.
- Pan American Allergy Society, San Antonio, Texas, March 14-17, 2002.
- Twentieth International Symposium On Man and His Environment in Health and Disease, Dallas, Texas, June 6-9, 2002.
- American Academy of Otolaryngic Allergy, San Diego, CA. September 19-21, 2002.
- International Symposium on Indoor Air Quality and Health Hazards, Tokyo, Japan. January 8-11, 2003.
- La Sociedad Mexicana de Alergia en Otorrinolaringología, Aguascalientes, Mexico. February 26-28, 2003.
- Pan American Allergy Society, San Antonio, Texas. March 20-23, 2003.
- American Academy of Environmental Medicine, Plano, Texas. April 3-7, 2003.
- Nutrition for Optimal Health Association, Inc., "Environmental Aspects of Health and Disease," Chicago, Illinois. May 7, 2003.
- 5th Congresso Internazionale Teorico Pratico di Nutrizione Olistica, Rome, Italy. May 23-25, 2003.
- 21st International Symposium on Man and His Environment in Health and Disease, Dallas, Texas. June 19-22, 2003.
- 62nd Annual Meeting, American Academy of Otolaryngic Allergy/Foundation, Orlando Grand Lakes, Florida. September 18-20, 2003.
- Chemical Injury Information Network, Fairfax, Virginia. October 5, 2003.
- American Academy of Environmental Medicine, Phoenix, Arizona. October 30 - November 2, 2003.
- La Sociedad Mexicana de Alergia en Otorrinolaringología, Aguascalientes, Mexico. February 25-28, 2004.
- Pan American Allergy Society, San Antonio, Texas. March 11-14, 2004.
- American Academy of Environmental Medicine, Overland Park, Kansas, April 15-19, 2004.
- IDEA 2004, Miami, Florida, April 27-29, 2004.
- The American Academy of Integrative Medicine, Manhattan, NY, April 30, 2004.
- 22nd Annual International Symposium on Man and His Environment in Health and Disease, Dallas, Texas, June 24-27, 2004.
- 63rd Annual Meeting, American Academy of Otolaryngic Allergy, New York, NY, September 17-20, 2004.
- 39th Annual Meeting, American Academy of Environmental Medicine, Hilton Head Island, SC, October 28-31, 2004.
- La Sociedad Mexicana De Alergia En Otorrinolaringología A.C., Veracruz, Mexico, March 2-5, 2005.
- Pan American Allergy Society, Grapevine, TX, March 17-20, 2005.
- American Academy of Environmental Medicine, Oakbrook, IL, April 13-18, 2005.
- Annual Raymer Family Lecture, Sir Mortimer B. Davis-Jewish General Hospital - McGill University, Montreal, Canada, April 28, 2005.
- 22nd Annual International Symposium on Man and His Environment in Health and Disease, Dallas, Texas, June 24-27, 2005.

William J. Rea, M.D.

- Mountain Valley Spring Company, Hall of Fame, Charleston, SC, October 7-8, 2005.
- 40th Annual Meeting, American Academy of Environmental Medicine, Tucson, AZ, October 27-30, 2005.
- Congreso Anual Internacional Sociedad Mexicana de Alergia en Otorrinolaringología, Mazatlan, Mexico, February 15-18, 2006.
- Intestinal Health...And Beyond, Dallas, Texas, March 3-5, 2006.
- Pan American Allergy Society, Grapevine, Texas, March 9 - 12, 2006.
- 13th International Symposium on Functional Medicine, Tampa, Florida, April 19-22, 2006.
- American Academy of Environmental Medicine, Kansas City, Kansas, April 27-30, 2006.
- American College for Advancement in Medicine, Dallas, Texas, May 5-6, 2006.
- Eighth Congress of Olistic Nutrition, Paestum, Italy, May 12-14, 2006.
- La Sociedad Mexicana de Alergia en Otorrinolaringología, Saltillo, Coah. Mexico, August 31 - September 2, 2006.
- Defeat Autism Now, Seattle, WA, October 6-8, 2006.
- American Academy of Environmental Medicine, St. Louis, Missouri, February 22-24, 2007.
- Pan American Allergy Society, Grapevine, Texas, March 15-18, 2007.
- Defeat Autism Now, Alexandria, Virginia, April 19-23, 2007.
- 25th Annual International Symposium on Man and His Environment in Health and Disease, Dallas, Texas, June 7-10, 2007.
- California Naturopathic Doctors Association, San Diego, California, October 20 - 21, 2007.
- American Academy of Environmental Medicine, Rancho Mirage, California, October 31 - November 4, 2007.
- American Academy of Environmental Medicine, Kansas City, Missouri, February 28 - March 2, 2008.
- Sociedad Mexicana Del Alergia En Otorrinolaringología, A.C., Saltillo, Coahuila, Mexico, March 5-8, 2008.
- II Congreso de Medicina Ambiental, Madrid, Spain, May 30 - June 2, 2008.
- 26th Annual International Symposium on Man and His Environment in Health and Disease, Dallas, Texas, June 19-22, 2008.
- Curso de Enseñanza en Alergia y Medicina Ambiental, Puebla, Mexico, September 3 - 6, 2008.
- 8th National Congress of the Italian Occupational and Environmental Allergic Dermatology Society (SIDAPA), Florence, Italy, October 23-25, 2008.
- American Academy of Environmental Medicine, Orlando, Florida, October 30 - 11/1/08.
- Pan American Allergy Society, The Woodlands, Texas, March 12-15, 2009.
- SOMAO Congress, Xalapa, Ver., Mexico, March 19-21, 2009.
- American Academy of Environmental Medicine, Overland Park, Kansas, April 2-6, 2009.
- Autoimmune Disease Symposium, San Francisco, California, April 22-26,

William J. Rea, M.D.

2009.

27th Annual International Symposium on Man and His Environment in Health and Disease, Dallas, Texas, June 25-28, 2009.

Congresso Internacional Sobre Medicina Ambiental, September 5-7, 2009, Manaus, Brazil.

Ontario Association of Naturopathic Doctors: Revolutionizing Medicine, The Connection Between the Environment and Health. November 13-15, 2009, Toronto, Ontario, Canada.

ABSTRACTS

1. Laseter, J.L., Rea, W.J., Buckley, T.P., DeLeon, B.S., Antoine, S.R.: Occurrence Of Chlorinated Phenoxy Acid Herbicides And Chlorinate Phenols In Environmentally Sensitive Patients. Presented, American Academy of Environmental Medicine. 1985.
2. Laseter, J.L., DeLeon, B.S., Antoine, S.R., Rea, W.J., Alger, C.: Analysis And Distribution Of Selected Volatile Organics In Whole Blood From Environmentally Sensitive Patients. Presented, American Academy of Environmental Medicine. 1985.
3. Jones, F.M., Butler, J.R., Lawlis, G.F., Rea, W.J.: Psychological Intervention Techniques: Part Of The Clinical Ecology Treatment Team Approach. Presented, American Academy of Environmental Medicine. 1986.
4. Sherek-O'Connor, R., Butler, J.R., Rea, W.J., Johnson, A.R.: Total Stress Load Inventory: A Validation Study. Presented, American Academy of Environmental Medicine. 1986.
5. Rea, W.J.: The Role of The Enzyme Detoxification Systems in Chemical Sensitivity.
6. Rea W.J., Ching, P.Y., Johnson, A.R., Butler, J., Laseter, J.: Organic Solvents - A Possible Etiology For Some Patient's Psychoimmunopathology.
7. Rea, W.J., Penyves, E.J., Johnson, A.R., Smiley, R.E., and Sprague, D.E.: A Double Blind Study of Chemical Sensitivity.
8. Rea, W.J.: Environmental Effects of ENT And Upper Respiratory System.

William J. Rea, M.D.

9. Rea, W.J.: Controlled Environment for Study of Environmental Pollutants in Buildings.
10. Rea, W.J., Smiley, R.E., Sprague, D.E., Johnson, A.R., de Victoria, A.L., Tucker, W.F., and Fenyves, E.J.: Bronchial Lavage as an Adjunct to the Treatment of Acute Recurring Asthma.
11. Rea, W.J., Smiley, R.E., Sprague, D.E., Johnson, A.R., de Victoria, A.L., Tucker, W.F., and Fenyves, E.J.: Chemical Sensitivity as a Result of Overexposure in The Work Place.
12. Rea, W.J., Smiley, R.E., Sprague, D.E., Johnson, A.R., de Victoria, A.L., Tucker, W.F., and Fenyves, E.J.: Formaldehyde Sensitivity Following Exposure to Building Material.
13. Rea, W.J., Smiley, R.E., Sprague, D.E., Johnson, A.R., de Victoria, A.L., Tucker, W.F., and Fenyves, E.J.: The Pancreas as a Target Organ in The Chemically Sensitive Individual.
14. Rea, W.J., Smiley, R.E., Sprague, D.E., Johnson, A.R., Edgar, R.T., Fenyves, E.J., Greenberg, M., and Williams, M.L.: Food Injection Therapy.
15. Rea, W.J., Smiley, R.E., Sprague, D.E., Johnson, A.R., de Victoria, A.L., Tucker, W.F., and Fenyves, E.J.: Bronchial Lavage as an Adjunct to the Treatment of Acute Recurring Asthma.
16. Rea, W.J., Smiley, R.E., Sprague, D.E., Johnson, A.R., de Victoria, A.L., Tucker, W.F. and Fenyves, E.J.: Chemical Sensitivity as a Result of Overexposure in the Work Place.
17. Rea, W.J., Smiley, R.E., Sprague, D.E., Johnson, A.R., de Victoria, A.L., Tucker, W.F. and Fenyves, E.J.: Formaldehyde Sensitivity Following Exposure to Building Material.
18. Rea, W.J., M.D., F.A.C.S., F.A.C.A.: Which Environmental Substances Can Cause Illness.
19. Rea, W.J., M.D., F.A.C.S., F.A.C.A.: Immunological and Non-Immunological Mechanisms.
20. Rea, W.J., M.D., F.A.C.S., F.A.C.A.: Management of Chemical Exposures.
21. Kuehn, K.A., Johnson, A.R., Rea, W.J.: Sequential Sampling and Identification of Fungal and Pollen Genera Within the

William J. Rea, M.D.

Dallas City Area: The Floral Picture?

22. Kuehn, K.A., Johnson, A.R., Rea, W.J.: Preliminary Observations on the Airborne Mycofloral Component Within the North Dallas metroplex.
23. Kuehn, K.A., Johnson, A.R., Rea, W.J.: Airborne Algae: Is It Prevalent? Is It Important?
24. Kuehn, K.A., Johnson, A.R., Rea, W.J.: Airborne Mycota Identified from Domestic Interiors Within Various Regions of Texas.
25. Kuehn, Kevin A., Johnson, Alfred R., Rea, William J.: Ascomycetes: An Overlooked Aeroallergen.
26. Kuehn, Kevin A., Johnson, Alfred R., Rea, William J.: Superficial Dermatomycotic Infection By Common Edaphic Fungal Species.
27. Kuehn, Kevin A., Rea, William J., Johnson, Alfred R.: Sequential Sampling and Identification of Pollen Genera Within The North Dallas Metroplex: Seasonal Variation Over A Three Year Period.
28. Chen, B., Price, S.C., Bridges, J.W.: How Environmental Chemicals May Contribute to "Total Body Load" - A Study of the Effect of Four Compounds on the Liver-Thyroid Axis.
29. Rea, W.J., Pan, Y., Johnson, A.R., Ross, G. H., Suyama, H., Fenyses, E.J.: Progress on Persian Gulf War Illnesses
30. Rea, W.J., M.D., F.A.C.S., F.A.C.A.: Mitogenic Effects of Mycotoxins on T₄ Lymphocytes.
31. Baird, N., Deborah, Rea, W.J.: The Temporomandibular Joint, The Implant Controversy.
32. Nicolson, Garth, L., Hyman, E., Both-Korenyi, A., Lopez, D.A., Nicolson, N., Rea, W., Urnovitz, H.: Progress on Persian Gulf War Illnesses - Reality and Hypotheses.
33. Higuchi, H., Miyata, M., Ishikawa, S., Rea, W. J.: Abnormalities of the Autonomic Nervous System in Chemically Sensitive Patients with Silicone Breast Implants.

William J. Rea, M.D.

34. Rea, W.J., Pan, Y., Fenyves, E.J., Griffiths, B: The Clinical Implementations of Autogenous Lymphocytic Factor in the Chemically Sensitive.
35. Rea, W.J., Griffiths, B.B., Griffiths, B., Pan: The Role of the Cell Cycle and An Autogenous Lymphocytic Factor In Clinical Medicine.
36. Mehra P, Rea W, MD, Wolford LM, DDS: Metal Hypersensitivity in Complex TMJ Patients.

POST GRADUATE TRAINING PROGRAMS

- I. From the Environmental Health Center - Dallas, affiliated with:
 - A. The University of Texas at Dallas, Ervin Fenyves, Ph.D.
 - B. The North Texas State University, Joel R. Butler, Ph.D.We have now graduated 10 individuals who received their Ph.D. from research done in collaboration with our Center.
- II. Clinical Training, M.D., D.O. - 9 months to 3 years. Programs - 10 individuals have completed.
- III. Research Fellowships from the Faculty of Medicine; Peking Union Medical School, Peking, China, 3 completed.
- IV. Each year we organize and sponsor The International Symposium on Man and His Environment in Health and Disease.
- V. We have an agreement with the Kitasato Medical School Department of Ophthalmology. Satoshi Ishikawa, M.D. is the Dean of Ophthalmology. We have trained 7 of his faculty in ophthalmology, each who have spent a year with us. We have done numerous research projects and published papers on the research.

IN THE MATTER OF
THE LICENSE OF
WILLIAM JAMES REA, M.D.

BEFORE THE
TEXAS MEDICAL BOARD

MEDIATED AGREED ORDER

On the 27 day of August, 2010, came on to be heard before the Texas Medical Board (the "Board"), duly in session, the matter of the license of William James Rea, M.D. ("Respondent").

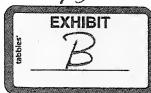
On November 16, 2006, Respondent appeared in person, with counsel Stephen A. Coke, at an Informal Show Compliance Proceeding and Settlement Conference ("ISC") in response to a letter of invitation from the staff of the Board. The Board's representatives were Keith Miller, M.D. and Paulette Southard, members of the Board. Mark Martyn represented Board staff.

Following the ISC a formal complaint was filed at the State Office of Administrative Hearings ("SOAH"). Subsequent to the filing at SOAH a mediation conference was held on August 21, 2006. Respondent appeared with counsel, Algis Augustine. The Board was represented Scott Freshour.

The matter did not settle at mediation. Respondent then retained Jacques Simon as lead counsel. Discovery was undertaken in this matter. After discovery was completed but prior to convening the contested case hearing the parties reached settlement.

BOARD CHARGES

Board Staff filed a complaint at the State Office of Administrative Hearings ("SOAH") charging Respondent with violations related to five patients. The charges concerned Respondent's diagnosis and treatment of "chemical sensitivity." After the completion of discovery, it appears that notwithstanding the allegations of the complaint, the primary concern of the Board relates to and focuses on Respondent's use of chemical antigens and the informed consent for such treatment.



BOARD HISTORY

Respondent has not previously received a disciplinary order from the Board. Upon the recommendation of the Board's representatives and with the consent of Respondent, the Board makes the following Findings and Conclusions of Law and enters this Agreed Order.

FINDINGS

The Board finds that:

1. Respondent received all notice required by law. All jurisdictional requirements have been satisfied. Respondent waives any defect in notice and any further right to notice or hearing under the Medical Practice Act, Title 3, Subtitle B, Texas Occupations Code (the "Act") or the Rules of the Board.
2. Respondent currently holds Texas Medical License No. D-2294. Respondent was originally issued this license to practice medicine in Texas on June 22, 1965. Respondent is also licensed to practice in Ohio, Arkansas, and Illinois.
3. Respondent is primarily engaged in the practice of environmental medicine. Respondent is board certified by the American Boards of Cardiovascular Surgery and General Surgery, members of the American Board of Medical Specialties.
4. Respondent is a member of the American Academy of Environmental Medicine and the Pan American Allergy Society, and practices medicine pursuant to the guidelines of those professional associations and has certifications from those medical professional organizations.
5. Respondent is 75 years of age.

Specific Findings:

1. The case involves five patients that were diagnosed with chemical sensitivity and/or environmentally sensitivity.
2. Respondent made these determinations based on use of various tests, including but not limited: SPECT brain scan, pupillography, thermography, heart rate variability, and intradermal skin testing for sensitivity to such things as: jet and diesel fuel, natural

gas, titanium, and lake algae. The intradermal testing was the primary concern of the Board related to testing because certain injections purported to be extracts of jet fuel and diesel fuel exhaust fumes and other chemicals. Respondent denied that the injections contained any harmful substances.

3. Respondent's treatment of these patients included: environmental controls; heat depuration therapy; intravenous therapies; oxygen treatments, and antigen injections. The antigen injections were the primary concern of the Board because certain injections purported to be extracts of jet fuel and diesel fuel exhaust fumes and other chemicals. Respondent denied that the antigens contained any harmful substances.

2. Respondent during his deposition of May 21, 2010 stated that there are no active chemicals in any of the chemical antigens, only the "electromagnetic imprint" of the chemical. Respondent testified that he uses in his testing and treatment of patients antigens containing electromagnetic imprint of the following: natural gas; propane gas; ethanol; formaldehyde; phenol; unleaded gasoline and jet fuel. Respondent testified that the antigens are in fact homeopathic remedies rather than substances containing actual chemicals. Respondent testified that none of the antigens are extracts of the actual substances specified in this paragraph.

3. Board staff asserts Respondent's treatment is unsupported by medical research and is non-therapeutic. In addition, Board Staff asserts there was a lack of proper informed consent for these treatments.

4. Respondent asserts that his diagnosis, care, and treatment of the above patients was appropriate and in accordance with established principles of medicine and peer reviewed articles disclosed to the Board.

6. Respondent admitted his current Informed Consent documents did not disclose that his antigen injections, were not FDA approved, and did not disclose that the chemical antigens mentioned in paragraph "2" above contained only the "electromagnetic imprint" of the chemical.

1. Mitigating Factors

- a. In determining the appropriate sanctions in this matter, the Panel considered the following mitigating factors:

- i. Respondent has cooperated in the investigation of the charges related to this Agreed Order. Respondent's cooperation, through consent to this Agreed Order, pursuant to the provisions of Section 164.002 the Act, will save money and resources for the State of Texas. To avoid further investigation, hearings, and the expense and inconvenience of litigation, Respondent agrees to the entry of this Agreed Order and to comply with its terms and conditions.
- ii. There were no claims of patient harm.
- iii. Respondent's patients continue to support him.

CONCLUSIONS OF LAW

Based on the above Findings, the Board concludes that:

1. The Board has jurisdiction over the subject matter and Respondent pursuant to the Act.
2. Section 164.051(a)(6) of the Act, as defined by Board Rule §190.5(I), failure to obtain informed consent from the patient or other person authorized by law to consent to treatment on the patient's behalf before performing tests, treatments or procedures.
3. Section 164.001 of the Act authorizes the Board to impose a range of disciplinary actions against a person for violation of the Act or a Board rule.
4. Section 164.002(a) of the Act authorizes the Board to resolve and make a disposition of this matter through an Agreed Order.
5. Section 164.002(d) of the Act provides that this Agreed Order is a settlement agreement under the Texas Rules of Evidence for purposes of civil litigation.

ORDER

Based on the above Findings and Conclusions of Law, the Board ORDERS that Respondent shall be subject to the following terms and conditions:

1. Respondent shall present the approved revised Informed Consent Form attached to this Order, to each and every patient who is undergoing or will undergo antigen

injections for chemical/environmental sensitivity ("Therapy"). Respondent shall include in the revised Informed Consent Form, written disclosures that explicitly state the following information:

- a. notice that the Therapy being offered is not FDA approved, and that this Therapy is considered non-traditional medicine (this notice shall be written in bold, oversized print);
- b. the effectiveness/therapeutic value of Therapy is disputed;
- c. a disclaimer that formulations prescribed have never been tested by the FDA for determination of the actual contents or the medical effectiveness;
- d. a written disclaimer that the "therapeutic value" of the Therapy, if any, has not been established or proven and is subject of dispute.
- e. The following Disclaimers shall be made all capital bold type:
 - i. **"THE TREATMENT/ANTIGEN THERAPIES BEING UTILIZED AND DESCRIBED BY RESPONDENT IN THIS DISCLOSURE STATEMENT DOES NOT CONTAIN ANY OF THE ACTUAL ACTIVE AGENT LISTED, AND CONTAINS ONLY "ELECTROMAGNETIC IMPRINT" OF THE AGENT. THE PATIENT IS NOT BEING INJECTED WITH ACTUAL ACTIVE AGENTS LISTED ON THE ANTIGEN"**

ii. "THE TREATMENT/ANTIGEN THERAPY BEING UTILIZED AND DESCRIBED BY RESPONDENT IN THIS DISCLOSURE STATEMENT IS NOT ENDORSED, SANCTIONED, OR SUPPORTED BY THE TEXAS MEDICAL BOARD."

2. Respondent shall be required to have each patient sign an acknowledgment. This acknowledgment is specifically applicable only to those patients receiving Therapy from Respondent and/or employees of his practice. The acknowledgement shall state that: on the initial and/or first visit, after the effective date of this Order, the patient received a written copy of the Informed Consent described in Ordering Paragraph No. 1.

3. Respondent must keep the signed acknowledgement in the medical record of each patient and an additional copy of each Informed Consent and signed acknowledgement in a separate file. This separate file shall be made available to the Compliance Division upon request to verify compliance with requirements of Ordering Paragraphs Nos. 1 and 3 above.

4. In addition, Respondent shall not start using any now Therapy, antigens, or other formulations that contain any amounts of the active ingredient of substances that are classified as hazardous substances and/or carcinogens by EPA, Agency for Toxic

Substance Registration & Disease Registry (ATSDR), OSHA, or any other federal or state regulatory agency.

5. Respondent shall not change, modify, or alter his current antigen protocol as provided to Board Staff and described during his deposition on May 21, 2010.

6. Respondent shall comply with all the provisions of the Texas Medical Practice Act and all other state and federal statutes regulating the Respondent's practice.

7. Respondent shall fully cooperate with the Board and the Board staff, including Board attorneys, investigators, compliance officers, consultants, and other employees or agents of the Board in any way involved in investigation, review, or monitoring associated with Respondent's compliance with this Order. Failure to fully cooperate shall constitute a violation of this order and a basis for disciplinary action against Respondent pursuant to the Act. Cooperation within the meaning of this agreement shall include providing Board staff or designees with samples of the antigens to be tested.

8. Respondent shall inform the Board in writing of any change of Respondent's mailing or practice address within ten days of the address change. This information shall be submitted to the Permits Department and the Director of Compliance for the Board. Failure to provide such information in a timely manner shall constitute a basis for disciplinary action by the Board against Respondent pursuant to the Act.

9. Any violation of the terms, conditions, or requirements of this Order by Respondent shall constitute unprofessional conduct likely to deceive or defraud the public, and to injure the public, and shall constitute a basis for disciplinary action by the Board against Respondent pursuant to the Act. Respondent shall be provided 30-day notice of a Probationer Show Compliance Proceeding to address any allegation of non-compliance of this Agreed Order as required by the Medical Practice Act

10. The above-referenced conditions shall continue in full force and effect without opportunity for amendment, except for clear error in drafting. If, after the passage of the 12-month period, Respondent wishes to seek amendment or termination of these conditions, Respondent may petition the Board in writing. The Board may inquire into the request and may, in its sole discretion, grant or deny the petition without further appeal or review. Petitions for modifying or terminating may be filed only once a year thereafter.

11. This Order resolves in their entirety the following board matters concerning Respondent: SOAH Docket No. 503-07-4032, and Investigative Log or case Nos. 10-4857 and 08-1434. The Board shall take no further action against the respondent with respect to the three matters referenced above and the Board's files regarding these matters shall be closed.

RESPONDENT WAIVES ANY FURTHER HEARINGS OR APPEALS TO THE BOARD OR TO ANY COURT IN REGARD TO ALL TERMS AND CONDITIONS OF THIS AGREED ORDER. RESPONDENT AGREES THAT THIS IS A FINAL ORDER.

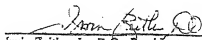
THIS ORDER IS A PUBLIC RECORD.

I, WILLIAM JAMES REA, M.D., HAVE READ AND UNDERSTAND THE FOREGOING AGREED ORDER. I UNDERSTAND THAT BY SIGNING, I WAIVE CERTAIN RIGHTS. I SIGN IT VOLUNTARILY. I UNDERSTAND THIS AGREED ORDER CONTAINS THE ENTIRE AGREEMENT AND THERE IS NO OTHER AGREEMENT OF ANY KIND, VERBAL, WRITTEN OR OTHERWISE.

DATED: 6-29, 2010.


WILLIAM JAMES REA, M.D.
Respondent

SIGNED AND ENTERED by the presiding officer of the Texas Medical Board on this
27 day of August, 2010.


Irvin Zeitler, Jr., D.O., President
Texas Medical Board

X. RELATED PROCEEDINGS APPENDIX

None.